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COMMUNICATIONS

LYMPHOID TUMOUR OF THE LACRYMAL GLAND

BY

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LYMPHOID growths of the lacrymal gland are perhaps sufficiently uncommon to make the following notes of a case of interest.

Catherine B., aged 9 years, was brought to the ophthalmic out-patient department at the War Memorial Hospital, Darlington, on May 28, 1937, by Major Russell, R.A.M.C., of Catterick Camp, and examined by one of us (A. McR.).

There was a history that the child had knocked the right eye against a radio set some six months before. No swelling developed at the time, and nothing abnormal was seen until three weeks before her appearance at the clinic when she was noticed to squint. Shortly afterwards a swelling in the right upper lid was seen. This had not increased in size since its first appearance. There was no complaint of pain.

An X-ray photograph of the skull taken on May 24 had not shown any bony abnormality.

The child was somewhat pale, but otherwise appeared healthy.

The right upper lid was in a condition of partial ptosis. Through it the sharply defined edge of an enlarged lacrymal gland could be felt projecting some 5mm. from the bony margin of the orbit. The skin of the lid was freely movable over the swelling and was slightly blue.

Vision in the right eye was 6/12 (partly): in the left eye 6/6 (most letters). There was no apparent squint and no diplopia could be elicited. The pupils were equal and reacted normally, and nothing abnormal was seen in media or fundi. No attempt was made to estimate any refractive error.

The opinion was expressed that the condition was due to a tumour of the lacrymal gland, and that operation would be necessary. She was given an iodide mixture for three weeks. On her return on June 18 the swelling was noted to be more pronounced and she was put on the waiting list for operation.

A day or two later while running in a children's race she fell and thereafter complained of pain in the left leg, and was sent into Hospital for observation on June 23. On the previous day she had an attack of epistaxis lasting two hours. For four days before admission she had vomited all her food.

She was admitted under Dr. G. F. Walker and most of the clinical notes below are from his records, which he has kindly placed at our disposal.

On admission further facts in her history elicited were that she had been born abroad (? India), had had malaria, measles, and whooping cough.

She kept the left hip flexed and cried on any attempt to straighten it. The upper part of the thigh was tender to touch. There was a hard swelling in the head of the left fibula fixed to the bone. (This was probably a haematoma as it disappeared during her stay in Hospital.) The left thigh was ½″ less in circumference than the right. Some tenderness was noted in the right iliac fossa; there were palpable glands in each groin. Her complexion was yellowish.

Blood:—Examination of the blood by the House Physician on several occasions revealed profound anaemia:—

	Hb.	R.B.C.	W.B.C.
June 26	40 per cent.	2,250,000	4,000
June 27	48 per cent.	1,520,000	3,200
July 1	46 per cent.	1,790,000	8,750
July 11	32 per cent.	1,150,000	3,100

On June 26 he noted anisocytosis and large red cells; on July 11 large numbers of malformed red cells, but no nucleated reds. A differential count on the latter date was as follows:—polymorphs 76 per cent., large lymphocytes 12 per cent., small lymphocytes 12 per cent.

Examination made by one of us (H.A.C.) on July 16 resulted as follows:—W.B.C. 2,950. Differential count:—polymorphs 69 per cent., lymphocytes 24 per cent., eosinophils 2 per cent., basophils 0 per cent., large mononuclears 5 per cent. Stained

films only suggest the picture of a megalocytic anaemia. Blood culture—sterile.

In view of the final diagnosis it should be stressed that the blood picture was never suggestive of a leukaemic condition. The white count was (except on July 1) very much below normal. And at no time were any immature white cells seen which might have suggested the diagnosis of an aleukaemic leukaemia. The blood picture was simply that of a secondary anaemia which might follow any recurrent loss of blood, *e.g.*, from epistaxis.

Other pathological examinations made by H.A.C. were:—

Widal:—Negative.

Wassermann:—Negative.

Throat swabs, right and left tonsils:—Streptococci, etc., not found.

Urine:—This contained albumen on one occasion (July 7). Acetone was twice found (August 15 and 24).

Temperature:—On admission the temperature was 97°F.; next morning it rose to 101° but fell to normal, and for the next week was not above 99°. Thereafter a condition of irregular pyrexia set in and continued till the end. Temperatures of 103° were several times recorded: in the last few days of her life 104° was twice reached, and on the day before her death 106°. She died on August 29.

Epistaxis:—This recurred twice during her stay in Hospital, and was severe on both occasions.

Course of the illness:—The left leg was fixed for a week or two in a Thomas's splint and gradually became less painful.

On July 10 two tender swellings appeared in the right thigh. These were apparently deep haematomata. They disappeared in the course of a week.

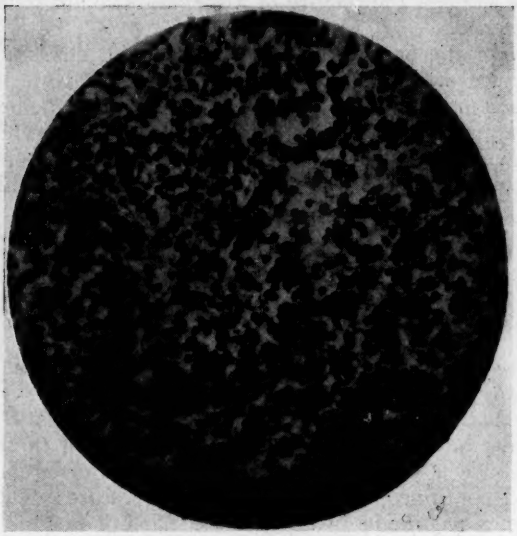
The swelling in the right orbit seemed at first to lessen in size, but subsequently returned to the size first noted. Only during the last few days of her life was there any increase in size apparent.

Its presence was over-shadowed by the anaemia and pyrexia, and until the post-mortem revealed the true state of affairs the diagnosis was uncertain. Malaria was thought of, but no parasites were found in the blood. Tubercle seemed to be the most probable solution of the problem.

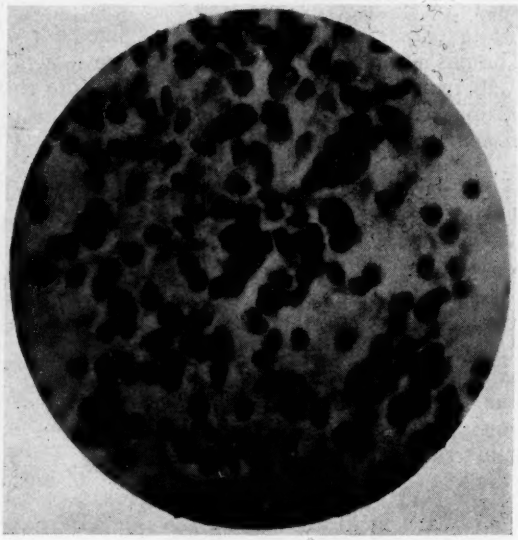
Blood transfusion from one of her parents was performed on two occasions, 8 ounces on July 16; 15 ounces on August 20. No lasting improvement resulted.

During the last few days of her illness petechial haemorrhages appeared in the skin.

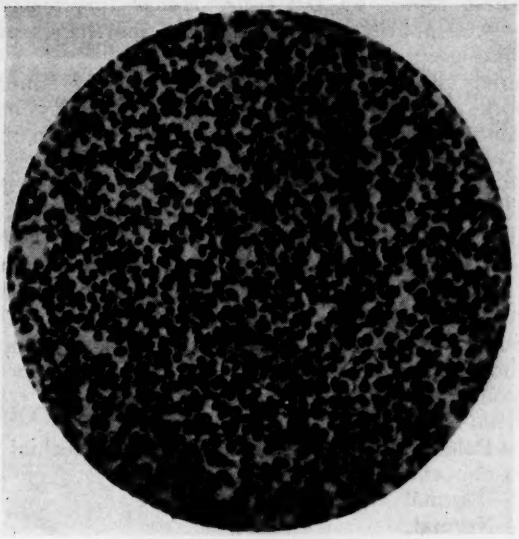
In view of the general symptoms removal of the enlarged lacrymal gland was never seriously considered.



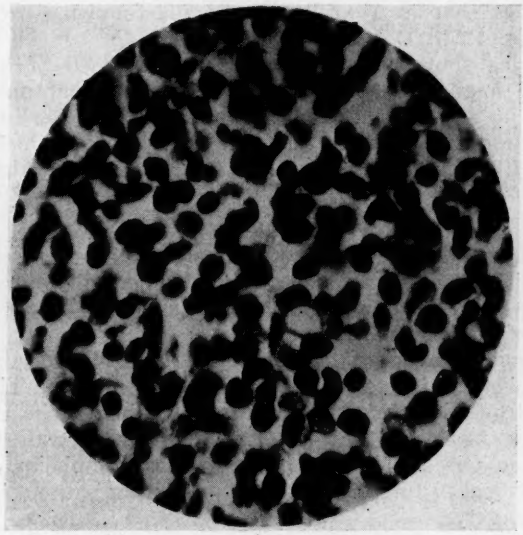
Lacrymal Gland. ×220



Lacrymal Gland. ×650



Gland from Abdomen. ×220



Gland from Abdomen. ×650

Post-mortem report by Dr. Gale, House Physician: "Body moderately well nourished. Petechial haemorrhages present on abdomen and legs; a few on arms.

On opening abdomen intestines were slightly adherent with fine net-work. Both large and small gut showed agonal spasm. No free fluid in abdomen. Small rubbery glands in mesentery. Gut pale; no haemorrhages in gut.

Spleen:—Enlarged: extended down to hand's breadth below ribs. Soft with no perisplenitis. Colour deep red. Nothing of note on section.

Liver:—Also greatly enlarged: petechial haemorrhages over its whole surface. Irregular areas of congestion seen on section.

Kidneys:—Normal size. Capsule not thickened. Stripped easily. Surface smooth apart from one area of scarring at upper pole of right opposite hilum. Cortex not diminished and of normal colour. Medulla and pelves normal.

Supra-renal:—Normal.

Heart:—Pale: normal size: covered with petechial haemorrhages: no abnormality on section.

Lungs:—Normal

Brain:—Normal.

Tumour of lacrymal gland:—Of same rubbery feel on palpation as glands found in mesentery."

Microscopic appearance of lacrymal gland:—The lacrymal tumour was sent to Moorfields, and the following report was received from Mr. Dee Shapland:

"Sections show that the tumour is composed of masses of large and small lymphocytes lying in a delicate fibrous stroma. There is no sign of encapsulation and the cells are infiltrating every portion of the tissues. Histologically the specimen is compatible with lympho-sarcoma or a leukaemic infiltration of the lid."

Summary

One of us (H.A.C.) has cut and studied further sections of the material from this case and also made micro-photographs from the same. Sections of a gland from the abdomen are very similar to those of the lacrymal tumour.

Lymphoid growths of the lacrymal gland may occur with or without characteristic changes in the blood count. This is referred to by J. S. Friedenwald in his Text Book "The Pathology of the Eye," p. 286. Further, Ewing in "Neoplastic Disease" refers to an observation made long ago by Cohnheim, viz., that the anatomical picture of leukaemia may occur without leukaemic blood changes.

The clinical and histological features in this case are suggestive much more of a leukaemic state than any other condition

and yet the blood picture does not help to confirm such a classification. Considering the question of lympho-sarcoma, it is true that such a growth may remain encapsulated for a considerable time, even for a year, but in this case there does not appear to have been any great increase in size of the growth and no evidence of any metastases. The glands, etc., in the abdomen found post-mortem would be in keeping, however, with the leukaemic theory. Further, H. P. L. Wells and M. S. Mayou, *Trans. Opthal. Soc. U.K.*, XXX, (1910), p. 97, point out that lympho-sarcomata of the lacrymal gland are usually met with in persons over 38 years of age.

It is, therefore, our belief that in this case the growth in the lacrymal gland was a leukaemic infiltration.

THE PHOTOCHROMATIC INTERVAL IN GLAUCOMA AND CAVERNOUS ATROPHY

BY

RANSOM PICKARD

EXETER

IN searching for some test which might prove useful in eyes which were possibly glaucomatous, a study was made of the field photochromatic interval. The results in glaucoma and cavernous atrophy seem worth communicating, in the hope that this test may be tried on a larger scale, further to test its usefulness.

In this paper the photochromatic interval will be abbreviated into " p.c.i." It is the condition in which a colour produces stimulus of light but not of colour. It can be produced in two ways; at a fixed spot by a minimal stimulus, which can be converted into a colour sensation by sufficient increase in brightness or enlargement of the area stimulated; or by movement in the field from without inwards to the fixation point. The area through which the colour stimulus is perceived as light, not colour, is the p.c.i. In this paper only the field is dealt with, and a moving object employed.

In the tests full daylight, varying from 150 to 240 foot candles at one metre, was employed with a Bjerrum screen as a background. " Ilford " coloured gelatin, mounted on pot opal, masked down to the various required sizes, was used. The opal was fastened to a flat piece of metal, from one end of which a bicycle brake wire, two inches long, was held on a Traquair's rod. By this means transmitted colours were used, not reflected.

Some preliminary experiments were made by the writer on his

right eye, to give data for comparison. They were made with the following colours:—

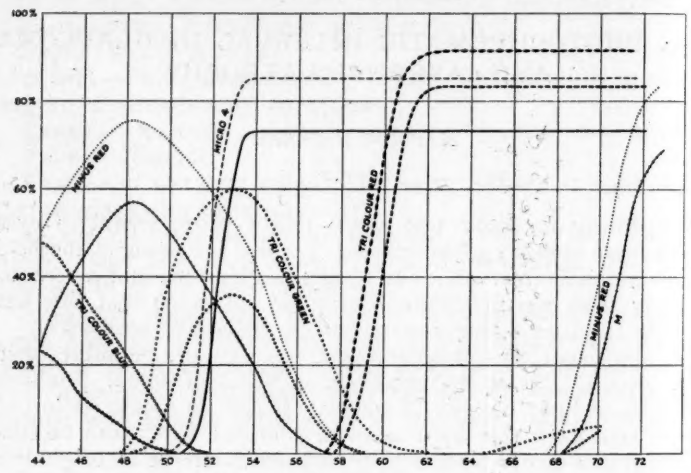
Ilford Tricolour—Green.
Ilford Tricolour—Red.
Ilford Micro 4 (Yellow).
Ilford Minus Red (Blue).

As used, in daylight, with the pot opal background, it is obvious that the daylight passed through the gelatines twice. By courtesy of the Ilford Company, a chart of the transmission

CHART A

Transmission data of some Ilford Gelatin Filters.

The transmission curves of the single filters are shown by dotted lines. The transmission curves for the doubled filters are show by full lines.

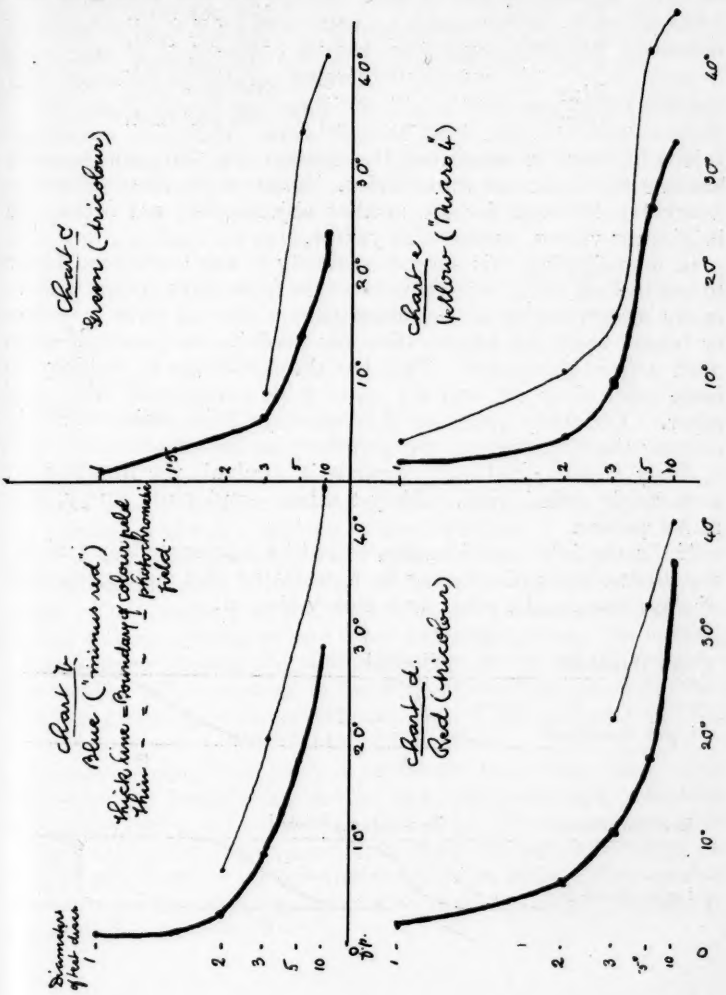


By permission of the Ilford Company, Ltd.

values for the four colours is shown. The double transmission colour values are:—

		Spectrum included	Excluded
	Per cent.		
Green - -	36	Green	Red, yellow
Red - -	83	Red	Blue, green, part yellow
Yellow - -	63	Yellow, red	Blue, green
Blue - -	57	Blue, part green	Yellow and red

The fields for circular discs varying from 1 to 10 mm. in diameter, the colours as above, were used to ascertain the normal photochromatic interval. The results are summarised in four charts (b—e). The average radii were ascertained by adding together the eight observations and dividing by eight. The abscissae represent the angles from the fixation point thus obtained. The co-ordinates are arbitrary values, representing the inverse of the diameters of the coloured discs multiplied by 10. Thus 1mm.=10, 1/10mm.×10=1, representing the sensitivities according to Weber's law.



It will be seen that, while the photochromatic interval persists with the yellow discs as far as the 1mm. disc, it is not so with the other colours. With the blue it ceases at the 2mm. disc, with green and red at the 3mm. disc. A comparison with the transmitted spectral values shows that the interval does not vary with these; for with the red having a transmission of 80 per cent. and the green of 36 per cent. the interval stops with the 3mm. disc.

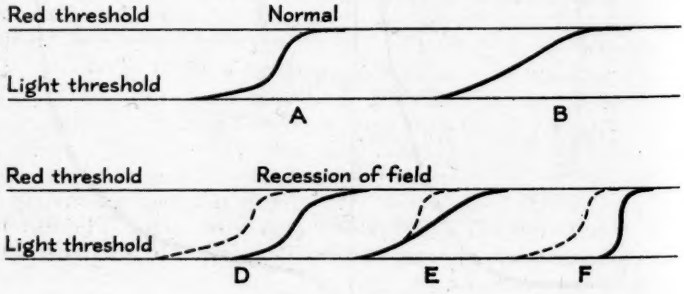
A brightness test in electric light, the filters being mounted on pot opal, thus giving double transmission as above, gave these results:—

Pot opal	100 per cent.
Yellow	100 per cent.
Red	46 per cent.
Blue	38·5 per cent.
Green	33 per cent.

It will thus be seen that the comparisons are not accurate because the brightness values differ. Even so, the photochromatic interval persistence for the smaller objects does not follow the brightness values, except with yellow.

In investigating this subject clinically it was considered better to use the red only, because the change from white to red is more easily recognised by an untrained person than of white to yellow or blue. Only the 10mm. disc was used in the group of cases dealt with in this paper. This has the advantage of making the issue more clear cut and the cases more comparable with each other. Generally speaking the transition from white to red in moving the objects from the periphery to the centre of the field is fairly sudden; but with some it is gradual, the intermediate zone being called pink, with yet others—and more rarely, it is called yellow.

If the rise into consciousness as red be represented by a slope, the sudden onset of red may be represented as a sudden increase of the slope A, the other as a steady rise, B.



On *a priori* grounds there are three obvious ways by which the photochromatic interval may be altered. Taking A as the commoner normal condition, D represents an equal diminution of perception for the p.c.i. interval and the red, E a diminution of the red field and increase of the p.c.i. interval, F a complete loss of the p.c.i. and slight alteration in the red field. Obviously these differences can be combined in various degrees, showing themselves as decreases or increases in the fields, either of the p.c.i. or red.

Opportunity has not offered itself to investigate the p.c.i. in sufficient cases of nervous or cerebral disease to make any report on them now, it is proposed to deal only with groups of glaucoma and cavernous atrophy at present.

The cases taken are consecutive and not selected, they are random in the proper sense because they came for examination without arrangement or collusion. It so happens that eighteen of each class occurred in the period of investigation. Dividing each group into two classes, those with a p.c.i. of under 5°, or 5° and over, the numbers are these :—

p.c.i.	Glaucoma	Cavernous atrophy
Under 5° - -	15	4
5° or over - -	3	14

The question at once arises whether this curious reversal in the classes in the two groups is mere chance, if another set of cases would have shown quite a different class grouping. This is possible but most improbable as the following arguments will show. The grouping of the p.c.i. into two classes is an example similar to coin tossing or any other two alternatives. In dealing with eighteen chances the possibilities are shown by the development of $(1+1)^{18}$, according to the arithmetical triangle of Jevons. Thus in grouping 3/15 the chances are 816/262,144 or 1 in 321; in 4/14 they are 3,060/262,144 or 1 in 85.

To supplement this method an actual experiment was made with coloured beads of equal size and spherical shape, amethyst and yellow, an equal number of each (41). These were mixed in a bag, a handful taken out and a bead dropped alternately in each of two cups till eighteen beads were in each. The number of each colour in each cup was then counted. In 162 counts the actual distribution was :—

TABLE I

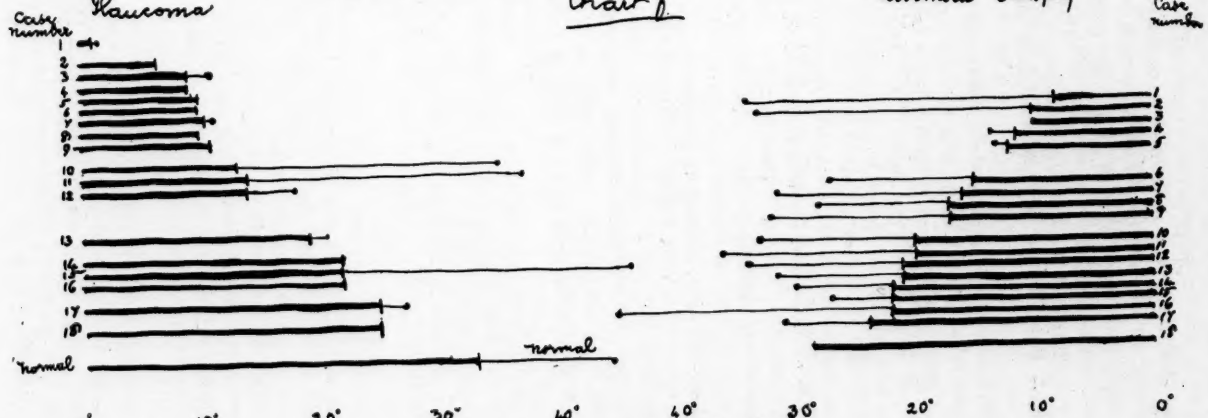
Probability Chart

Amethyst	Yellow	Actual Grouping (162 counts)	Theoretical Distribution $(1+1)^{18}$
0	18	–	1
1	17	–	18
2	16	–	153
3	15	1	816
4	14	1	3060
5	13	12	8568
6	12	18	18564
7	11	23	31824
8	10	35	43758
9	9	33	48620
10	8	16	43758
11	7	8	31824
12	6	2	18564
13	5	4	8568
14	4	–	3060
15	3	1	816
16	2	–	153
17	1	–	18
0	18	–	1
		Total	$262144=2^{18}$

Giving 1 count of the A15/Y3 group and 1 of the A3/Y15 group in 162 trials, 1 and 0 in the 14/4 groups. But the probability is much less than this. For the coincidence of the 15 amethyst 3 yellow with the 4 amethyst 14 yellow would be far less probable than the occurrence of either group with some other combination. Thus the two groups considered together are so improbable as a matter of chance that this may be neglected. They may be dealt with as due to some cause or causes other than chance.

In the chart " f " and Table II the cases are arranged in order of size of the red field, as found by a 10mm. disc of Ilford Tricolour red mounted on pot opal. It will be seen that of the glaucoma cases, 12 are 14° or less, 6 being spread out between

Red fields & Photochromatic intervals.
Chart I.



Thick lines = average radii red fields. from fixation point
Thin lines = photochromatic interval. (p.c.i.)

TABLE II

Glaucoma					Cavernous Atrophy				
Case No.	White Field 3/1000	White Field 1/1000	Red Field 10/1000	Photo-chromatic Interval	Case No.	White Field 3/1000	White Field 1/1000	Red Field 10/1000	Photo-chromatic Interval
1	10°	0	1°	0·5°	1	34	18	8	26
2	38·5	11·5	6·5°	0	2	33	4	10	23
3	38	19	9	2	3	36	16	10	0
4	22	5	9	0	4	30	11	10·5	2·5
5	22	10	10	0	5	38	22	12	1
6	10	7·5	10	0	6	46	21	15	12
7	39	19	10·5	0·5°	7	46	17	16	15·5
8	36	10·5	10	0	8	38	22	17	11
9	35	14	11	0	9	35	20	17	15
10	34	12	13	22	10	29	15·5	20	13
11	37	14·5	14	23	11	28	10·5	20	16
12	22	12	14	4	12	33·5	3·5	21	13
13	36	12	19	1·5	13	36	12	21	10·5
14	27·5	15	22	0	14	30·5	12	22	9
15	34	21·5	22	24	15	32	19	22	5
16	32	13	22	0	16	38·5	19	22	21
17	36	11	25	2	17	28	15	24	7
18	33	13	25	0	18	36	9	29	0
Average	29·7	12°	14°	4·6°	Average	34·8°	14·8	17·6	11·3
Standard Deviation	9·2	—	6·7	8·27	Standard Deviation	4·5	—	4·1	7·9
Probable Error	±2·23	—	±1·58	±2·25	Probable Error	±1·12	—	±0·99	±1·86

19° and 25°. The cavernous atrophy group shows 6 cases of 15° or less, 11 between 16° and 26°, and 1 of 28°. But the marked feature of the photochromatic interval in the glaucoma group is that it is absent or slight in every case but three, whereas it is present and large in all the cavernous atrophy cases except four, and in one of these four the red field is normal; if allowance is made for the variation in the normal red field that actually occurs.

Moreover, the chart shows that, if the red field and the p.c.i. be added together, the total field in the cavernous atrophy cases is not far short of the normal. In other words, these cases are examples of the " E " diagram method of decrease of the red field, the outer boundary of the p.c.i. being less affected than the red boundary. It will be seen that with the three exceptions in one group and four in the other, this distinction occurs throughout the whole series, irrespective of the size of the red field, an important point. The groups are in this respect curiously the converse of one another. Although this distinction is not absolute, there being the exceptions named in each group, it is sufficiently frequent to be a notable difference. That there are exceptions makes it more difficult to apply this rule to any particular case; but it may fairly be said that, if the red field is diminished and the photochromatic interval is under 5°, there is a five to one chance that it is glaucoma. Conversely, if the red is reduced and the p.c.i. is over 5°, the chances are one to four that it is cavernous atrophy.

The question as to whether the two groups, glaucoma and cavernous atrophy, are of comparable severity is important. As 3/1,000 white and 10/1,000 red give nearly the same field, if the p.c.i. be excluded, it is proposed to use the 3/1,000 white field as a standard of comparison. The full details are given in Table II.

It will be seen that the glaucoma 3/1,000 white field gives an average of 29·7°, cavernous atrophy of 34·8°, a difference of 5·1°, not great but decided. Thus on the whole the glaucoma series is more severe than the cavernous atrophy group. Yet it will be seen on inspection of the chart that the small or absent p.c.i. is well scattered over the whole of the glaucoma group, it is not confined to the severe cases. In the cavernous atrophy group the absent p.c.i. occurs in those with nearly average 3/1,000 white field.

The average of the red fields shows a difference of 3·6° between the two groups, which is almost within the range of patients' errors. But the p.c.i. is 4·6° in the glaucoma cases as against 11·3° in the cavernous atrophy group, a very marked distinction.

In clinical work we are often confronted with the problem

whether an eye is affected by glaucoma or cavernous atrophy, or whether it is healthy. Tension, white fields and central vision may be normal when examined. This question becomes more urgent when one eye has, without question, glaucoma, possibly in an advanced stage. Such a case was No. 9 in the glaucoma series; but the red field was down to 11°, with no p.c.i. There was additional confirmation of affection of this eye in a slightly lowered central light threshold (·0001875 ft. candles) and in the presence of two white fragments adherent to the pupil margin. The other eye had advanced glaucoma lento-exfoliativa.

It is easier to state the facts than to give an explanation of these alterations in the photochromatic interval. As the clinical defects do not go further back than the optic nerve, the explanation must allot the cause between the retina and the conducting fibres. In glaucoma the retinal visual elements are directly affected by pressure and so are the fibres, in the retina and the optic nerve head. In cavernous atrophy there is no pressure on the retina, whereas the nerve fibres are involved in the contraction of the nerve head. It would seem that pressure on the retina extinguishes the p.c.i. portion of the red field, as though that part of the retina were more susceptible to pressure than the area where red is seen as colour. Now the latter corresponds fairly closely to the 3/1,000 white field, which is found clinically to stand pressure in glaucoma better than the 1/1,000 or the 5/300 white field. But this explanation would be valid only if the red field remained of normal size, which is not the case in the glaucoma series. Therefore, the more general explanation must be adopted that pressure quickly obliterates the liminal subchromatic red sensation in whichever part of the retina this may be. The exceptional three glaucoma cases must be taken as indicating the possibility of occasional great powers of resistance.

Of the four exceptions in the cavernous atrophy group, one has a practically normal colour field. The obvious explanation of the other three is that they were near the 10° red field limit, within which the p.c.i. soon disappears. Yet it will be seen that cases 1 and 2 in this series have a large p.c.i. although the red fields are 8° and 10° respectively.

It would seem that more experience is required with this test, in cases where there is known to be nerve interruption, before any adequate explanation can be given.

In conclusion, this test of the size or absence of the photochromatic interval, is put forward as worthy of further investigation, in the hope that it may be of use, particularly in the early stages of glaucoma.

EXPERIMENTAL DATA ON THE PROBLEM OF THE PERMEABILITY OF THE CORNEA

I.—Experiments on Excised Pigs' Eyes and Living Rabbits' Eyes with Iodide and Nitrite

BY

DR. M. KLEIN

BUDAPEST

TOWARDS the end of the past century evidence had already been brought forward showing that the cornea plays an important rôle in the liquid exchange in the eye. Laqueur performed experiments which showed that potassium cyanide is capable of diffusion from the aqueous humour outwards through the cornea; he accorded the cornea an important rôle in the fluid exchange of the eye. Since that time this fact has been the subject for a good deal of discussion. The question of the permeability of the cornea is very important when it is considered that the greater part of all drugs applied to the conjunctiva enters the interior of the eyeball through the cornea. Nakamura, who investigated the question more thoroughly, showed that hyper-and hypotonic solutions diffuse rapidly through the cornea, while isotonic solutions do not do so. His opinion was that hypotonic solutions are more effective, for in this case the flow is from the outer surface to the inner which aids the diffusion of the substances dissolved in the liquid in question. According to the determinations made by Gallenga the hypertonic solutions diffuse relatively more rapidly. Antonibon considered the osmotic pressure differences a decisive factor in the permeability of the cornea and at the same time called attention to the fact that certain chemicals such as KCl, LiCl, increase the effectiveness of eye drops (cocaine, atropine, eserine, pilocarpine). In the presence of rubidium and caesium chlorides this effect is not noticeable. These examinations were made on living or excised animal eyes.

According to Fischer the cornea may be regarded as a membrane which hardly allows water to diffuse, while chloride ions diffuse in the epithelial-endothelial direction with difficulty and not at all in the reverse direction. Some of these results were obtained with eyes in contact with, or after being rinsed with sodium citrate, and some after the eye had been exposed to the influence of a concentrated saccharose solution. He determined the diffused chloride ions and analysed the aqueous humour. He found that epi-and endothelial lesions increased the original permeability at least ten times. It is apparent, therefore, that the permeability

is regulated by the presence and condition of these two layers. While these layers are anatomically and physically (as far as the swelling " Quellung " is concerned) in a physiological condition, diffusion takes place away from the layer more inclined to swell in the direction of the layer less able to swell, *i.e.*, in the epi-endothelial direction. Similar irreciprocal permeability was noticed by Wertheimer in the frog's lungs and skin, and by Cohnheim in the intestinal wall of the frog. Following this Fischer's experiments dealing with the swelling ability of the epi-and endothelial walls were of great importance. These experiments in which the ultramicroscope was employed proved that the epithelium is able to withstand less diluted solutions, retaining its original degree of swelling, than is the endothelium. His experiments with stains also showed that permeability is dependent on the condition of swelling of the corneal layers. A further observation was that acid stains, if finely dispersed, pass through the cornea, while basic stains do not.

Ridley (1930) described a series of experiments in the living eye which show that the cornea is permeable in both directions and that this permeability is most marked at the periphery at the site of the filtration angle. His experiments led to the conclusion that Descemet's membrane is relatively impermeable, but that the area described as the filtration angle is freely permeable to dissolved substances which spread rapidly by a process of diffusion throughout the substantia propria. He found that the epithelium is much less permeable than the substantia propria but demonstrated that it is permeable in both directions.

Author's Experiments

The purpose of these experiments was to examine the permeability of the cornea. The experiments were done with potassium and sodium iodide and sodium nitrite. The iodide experiments were done in two groups, in the first experimental series the determinations were made on freshly enucleated pigs' eyes, the second on living rabbits' eyes.

The pigs' eyes were placed on wooden slats which had a slight indentation in them, so that the cornea faced upward and a 1cm. diameter glass tube was placed on the cornea and held in place by a spring with slight pressure. The end of the glass tube was first rounded in a flame to prevent injury to the epithelium of the cornea. The edge of the glass tube was covered with vaseline to prevent the escape of the solution in the tube. In the " inside-out " determinations the experimental solution was placed in the anterior chamber through the optic nerve with a long syringe

needle, the needle was then withdrawn and the optic nerve tied off with a ligature.

In the experiments on living rabbits' eyes, the rabbits were under urethane anaesthesia and, with the head properly fixed, a glass tube 1/2cm. in diameter was placed on the eye. In the "inside-out" experiments a needle was used in the filling of the anterior chamber. To avoid loss of aqueous humour the needle was forced through the opposite side and was left in place for the duration of the experiment.

The iodide was determined by Scheffer's method, which fundamentally consists of the following procedure: in the presence of organic substance after the addition of KOH and oxidation in a sand bath, the oxidised product is extracted with alcohol, the iodine shaken out with carbon tetrachloride, iodated with chlorine water and determined titrametrically.

The determination of the nitrite was made with the colorimetric micromethod. In the first series of experiments made on pigs' eyes, potassium iodide was used. The outer solution placed in the tube was 2ccm. of 0·9 per cent. NaCl.

TABLE I

Pigs' eyes, in-out. Fluid in the tube 2 c.cm. 0·9 per cent. NaCl, tube diameter 10 mm.

No.	Into the A.C.	Time	Permeated
		minutes	
1	0·5 c.cm. 5 per cent. KI	60	202 γ
2	0·5 „ 5 „ „	60	210 γ
3	0·5 „ 2 „ „	60	100 γ
4	0·5 „ 2 „ „	60	74 γ
5	0·5 „ 2 „ „	60	56 γ
6	0·5 „ 2 „ „	75	104 γ
7	0·5 „ 2 „ „	75	144 γ
8	0·5 „ 2 „ „	120	204 γ
9	0·5 „ 2 „ „	120	240 γ
10	0·5 „ 2 „ „	120	182 γ
11	0·5 „ 2 „ „	120	99 γ

As the table shows iodine was demonstrable in quite large quantities in the outer solution.

TABLE II

Pigs' eyes, out-in. Into the A.C. 0·9 per cent. NaCl. In the tube 2 c.cm. 2 per cent. KI.

No.	Time	Aqueous	Permeated
	minutes		
1	60	0·3 c.cm.	130 γ
2	60	0·3 „	139 γ
3	60	0·3 „	147 γ
4	120	0·5 „	240 γ
5	120	0·5 „	345 γ
6	120	0·5 „	260 γ

In the second experimental group "outside-in" iodine diffusion was examined. The anterior chamber was filled with 0·9 per cent. NaCl solution and 2cm. 2 per cent. KI was placed in the tube.

As the table shows iodine was demonstrable in large quantities in the aqueous humour. As in the first group of experiments the permeated iodine increased proportionally with the time of the experiment.

In further experiments it was investigated whether or not the addition of 10 per cent. of blood-serum to the sodium chloride solution, acting as a colloid osmotic factor, increased the amount of diffused iodine.

TABLE III

Pigs' eyes, in-out. Into the A.C. 0·5 c.cm. 2 per cent. KI. Tube 0·9 per cent. saline +10 per cent. serum.

No.	Time	Permeated
	minutes	
1	60	92 γ
2	60	138 γ
3	60	77 γ
4	60	131 γ
		control 33 γ
		„ 107 γ

As control serve also the experiments, Table I, Nos. 3, 4, 5.

As is readily noticed, in the serum experiments the quantity of diffused iodine is increased.

The question was also investigated as to what extent the time elapsing since death affects the cornea with regard to its permeability.

TABLE IV

Pigs' eyes, in-out. Into the A.C. 0·5 c.cm. 2 per cent. KI. Tube 2 c.cm. 0·9 per cent. NaCl.

No.	Time since death	Time of experiment	Permeated
		minutes	
1	8 hours	150	460 γ
2	2 „	150	150 γ
3	2 „	60	223 γ
4	2 „	60	184 γ
3 and 4 the cornea was dipped in boiling water for 10 secs.			
5	2 hours	60	268 γ
6	2 „	60	307 γ
7	2 „	60	294 γ
5, 6 and 7 the cornea was dipped in boiling water for 30 secs.			
8	Control cornea not dipped in boiling water	60 minutes	115 γ

While very little iodine diffused through the freshly excised cornea, after standing in the refrigerator for 8 hours the diffused iodine increased almost 3 times the original amount. In a further experiment the eyeball was held with tweezers and only the cornea was dipped into boiling water. As is seen the adverse effect of 10 seconds doubly increased the quantity of diffused iodine, and after 30 seconds the quantity of demonstrable iodine was even greater.

Rabbit Eye

The experiments were performed in such a manner as to place the chemical to be examined in the aqueous humour without injury to the eye itself. This was done by giving the rabbit intravenous injections of 5 per cent. sodium iodide and allowing time for sufficient concentration of the iodine in the aqueous humour in order for it to diffuse to the outside surface. The animals received 6-10c.cm. according to body weight. During the experiment 34 γ of iodine was found in 1/10c.cm. of the aqueous humour. After an experimental time of 40 minutes

permeated iodine was not demonstrable in the outer solution. This is to be expected if we take into consideration the results of the former experiments where the diffused ion quantity is approximately 1·5-2 per cent. In this instance the amount of diffused iodine to be expected is so small as to be at or below the limit of demonstrability. Therefore in order that the small iodide concentration might diffuse through the cornea in sufficient quantity to be demonstrated, 6 hour experiments were done. The rabbit was under urethane anaesthesia during the experiment. During the experiment 2 intravenous injections were given, each 7ccm. of 5 per cent. NaI. The outer liquid was 0·9 NaCl solution. The second injection was given for the purpose of ensuring a proper blood iodine mirror. The liquid in the ring on the cornea was changed hourly. Iodine was demonstrable in the outer solution collected over the six hour period. The pink colour of the carbon tetrachloride corresponded, according to approximate estimations, to 5-7 γ of iodine.

In the case of intra-ocular injections of NaI, KI, the results were as follows:—

TABLE V

Rabbit eyes. In-out.

No.	In the a.c.	Time	Permeated iodine
		minutes	
1	0·3 c.cm. 5 per cent. NaI	15	202 γ
2	0·3 „ 5 „ „	15	93 γ
3	0·3 „ 5 „ „	15	284 γ
4	0·3 „ 5 „ „	15	214 γ
5	0·3 „ 5 „ „	15	216 γ
6	0·3 „ 2 „ KI	60	58 γ
7	0·3 „ 2 „ „	15	18 γ
8	0·3 „ 2 „ NaI	15	51 γ
9	0·3 „ 2 „ „	15	34 γ

As the table shows the diffused iodine was demonstrable after 15 minutes. After the intra-ocular injection of sodium iodide the cornea became a little hazy after 3 or 4 hours, but became clear again after a longer period of time. In many of the rabbits after the disappearance of the haziness a very fine nebula could be found.

It can be seen that iodine diffusion in living rabbits' and excised

pigs' eyes is quite possible in both directions. In these experiments not only the qualitative but the quantitative determinations of iodine were done.

Nitrite Experiments

On the pigs' eyes: the aqueous humour was replaced with 1/10 equimolar nitrite—Locke-solution /0·5c.cm./. The outer fluid was 0·9 per cent. NaCl solution.

TABLE VI

Pigs' eyes. Nitrite. In out.

No.	*Time*	Permeated iodine
	minutes	
1	60	5·18 γ
2	60	4·66 γ
3	60	6·78 γ

The experiments all show that nitrite is capable of diffusion through the cornea. The question of nitrite diffusion through the living rabbit cornea was also investigated. After the drainage of the aqueous humour 0·3c.cm.—Locke solution—was injected, of which 1/10th part was the above-mentioned nitrite solution. During the 80 minute experimental period, using a 5mm. diameter external collecting tube, 3·45 γ permeated. After a 15 minute experimental period the diffused nitrite amounted to 0·87 γ. In the aqueous humour, after the end of the 80 minute experimental period, only 4·9 γ of nitrite was demonstrable. These experiments show that the cornea is also permeable to nitrites.

Summary

In two groups of experiments it was shown that freshly enucleated pigs' eyes are equally permeable from " outside-in " and " inside-out " to KINaI and $NaNO_2$. The amount of salt recovered after passing through the cornea is closely proportional to the concentration of the salt and to the time elapsed in each case. Further experiments show that the permeability increases three times by the end of the 8 hour period after death, but is not materially increased during the first 2 hours from which the above observations were obtained. The addition of 10 per cent. of serum to the fluid applied to the epithelium increased the amount of salt permeating in one hour as compared with the control. By dipping the cornea into boiling

water for 10 and 30 seconds, injuring the epithelial cells and repeating these experiments, it was shown that destruction of the epithelium increases the amount of salt permeating.

A series of experiments on living rabbits under urethane anaesthesia using 5 per cent. and 2 per cent. concentrations of sodium iodide and 1/10 equimolar nitrite gave similar results. The amount permeating being approximately proportional to the concentration of the solution and the duration of the experiment. It is important to point out that if a known quantity of a dissolved salt is introduced into the anterior chamber of a living animal and the salt diffusing through the cornea is collected, it is found that only a small proportion, about 1·5-2 per cent. is recovered. This is due to the fact that the salt diffuses by other ways into the capillaries of the iris and ciliary processes from whence it is at once carried away by the blood stream, and to a less extent into the lens and vitreous humour. In control experiments it was found that at the end of 80 minutes 2 per cent. of the salt introduced had permeated but that only 3 per cent. could be recovered from the anterior chamber. It seems that for every 100 units by weight introduced into the anterior chamber 2 pass out through the cornea, 3 remain in the anterior chamber and 95 units are eliminated by diffusion in other directions in 80 minutes.

The objection may be raised against these experiments that both the iodide and nitrite are not found under normal conditions in the system and the possibility remains that the cornea is permeable to them as foreign substances, and that the cornea shows an entirely different behaviour to the ions normally found in the system. The question might also be raised whether concentration of the chemicals used was not abnormally high, and it might be held that the experimental results since they were obtained with hypertonic solutions, cannot be generalised.

It was necessary therefore to repeat the experiments with a special apparatus with ions normally found in the system in a physiological concentration or one close to that and further, to investigate the diffusion of water. These results obtained in collaboration with Dr. Sárkány are given in a separate paper.

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EXPERIMENTAL DATA ON THE PROBLEM OF THE PERMEABILITY OF THE CORNEA

II.—The Permeability of the Excised Cornea with Respect to Water and Chloride Ions

BY

DR. M. KLEIN *and* DR. J. SÁRKÁNY

THE data in the literature dealing with the permeability of the cornea give variable and dissimilar results both with regard to the irreciprocity of the permeability and to the magnitude and quality of the necessary osmotic forces and to the rate of diffusion. The purpose of our experiments was to estimate these discrepancies using a different method, and to examine the points brought out above.

Method:—Our experimental apparatus was similar to the chamber system described by Hamburger. The chambers were separated by the excised cornea. Various solutions were placed in the two chambers and the changes in volume, and in the chloride ion concentration were determined. The volume change was determined by capillary measurement, the chloride ion by a micro-method. (Diameter of capillary 0·8mm. 10 units of the capillary scale equalled 5c.mm.) The volume of each chamber was approximately 4c.cm. The solutions used were pig's blood diluted with Ringer's solution to various degrees. The air bubbles in each section were carefully displaced and liberated. The corneae were obtained with the aid of a slice made through the equator of the

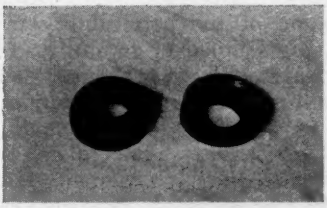

FIG. 1.

Moulded rings of soft rubber used to clamp the cornea.

globe and they were fixed between rubber rings at the scleral margin. (See Fig. I.) Usually three chambers and two corneae were used in such a manner that each experiment gave two parallel determinations.

Experimental Results

In the first instance we determined whether or not, with the aid of osmotic pressure, fluid would pass through the cornea. A photograph of the apparatus is given below.

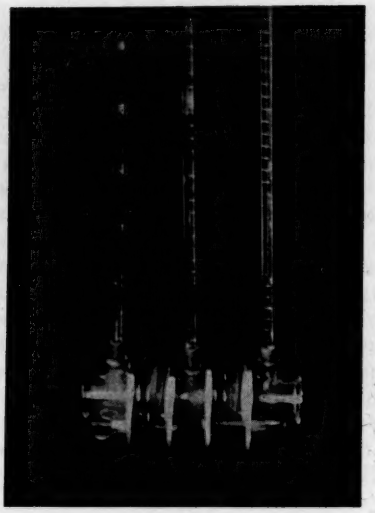

FIG 2.

The complete apparatus employing two corneae (epithelial or endothelial surfaces opposed).

In the determination of the permeability in the endothelial-epithelial direction, pig's blood or serum, or a dilution of these with Ringer's solution was placed in the outside chambers, and in the middle chamber there was Ringer's solution.

TABLE I

Flow of liquid effected by osmotic forces. The convexity of the sign ⊐ or ⊏ corresponds to the epithelial surface.

	Mins.	Blood	⊐	Ringer	⊏	Blood
Feb. 23, 1937	0–30	13		−7		10
	30–60	33		0		36
Feb. 23, 1937	0–30			−3		11
	30–60			0		28

		Doubly diluted serum	C	Ringer
Feb. 26, 1937	0–30	−3		−17
	30–60	15		3
	60–90	13		0

		Doubly diluted serum	C	Ringer	D	Doubly diluted serum
March 8, 1937	0–30	25		0		5
	30–60	5		0		5
	60–90	20		0		15
	90–120	25		0		15
	120–150	20		10		15
	150–180	10		5		10

		Doubly diluted serum	C	Ringer
March 5, 1937	0–30	−2		−15
	30–60	17		−12

		Doubly diluted serum	C	Ringer	D	Doubly diluted serum
March 8, 1937	0–60	−10		0		0
	60–90	10		0		15
	90–120	15		0		30
	120–150	20		5		25

		Doubly diluted serum	‖ †Ringer	D	Doubly diluted serum
March 18, 1937	0–20	−20	0		15
	20–40	−2	0		15
	40–60	0	0		15
	60–80	−5	0		5

‖ † Represents an impermeable wall.

Our results all showed that the volume of the solution containing a colloid was increased. The volume of the Ringer's solution either decreases or remains unchanged. In fact, in a few cases, in the later stages the volume of Ringer's solution increases, but this increase does not compare with the volume increase of the colloidal solution. This may be explained on the supposition that an outside factor (temperature change) influences the meniscus in all cases to the same extent. This possibility was, however, excluded by our experiments in which we determined the effect of this factor in a closed chamber of solution connected with a capillary, and even after taking this factor into consideration we discovered incongruous volume changes (Table I. III/18, 1937).

The explanation may be that the solution did not pass through the cornea, but the cornea itself exuded or absorbed fluid from

its surroundings. For instance a gel may take up or give off a measureable quantity of water without appreciable volume change. If this is the explanation the volume of the colloidal solution due to the effect of the cornea placed in it, as it comes in contact with both surfaces of the cornea, undoubtedly and measurably should increase. According to our experiments this does not occur, for a volume change is not appreciable.

TABLE II

The volume-changes of the cornea under the influence of various solutions.

	*Mins.	Doubly diluted serum +cornea	‖†Ringer	‖† Doubly diluted serum +cornea
March 10, 1937	0–60	5	0	0
	60–120	0	0	5

	**	Ringer +cornea	‖† Doubly diluted serum	‖† Doubly diluted serum +cornea
April 12, 1937	0–30	−10	−5	0
	30–60	0	5	5
	60–90	5	0	0

* The experiment was begun one hour late due to technical difficulties (*i.e.*, three hours after death).
** The experiment was begun half-hour late due to technical difficulties.
‖† Represents an impermeable wall.

We must therefore investigate a further supposition, that the changes noticed are brought about by the liquid passing through the cornea. The absence of a quantitative connection may be explained by the fact that, besides the diffusion, swelling of the cornea takes place under the influence of the various solutions, in such a manner that the results obtained are the resultant of the two changes occurring simultaneously. As to the acceptability of this explanation our experiments with chloride ions served as a basis which as we shall later show gave similar results.

We examined the diffusion in the epithelial-endothelial direction by placing the solutions in the appropriate chambers. The results (Table III) while coinciding with the former were not as constant, although it was to be expected from the data in the literature that a diffusion in this direction (epi-endothelial) could more easily be demonstrated. These experiments showed in most cases that in the first half of the experimental time the volume of the colloid solution increased, while the volume of the neighbouring chambers showed a variety of changes. In the second half of the experimental time in most cases the volume of the Ringer's solution increased, in some cases this was noticeable from the very beginning.

TABLE III

Flow of liquid through the cornea.

	*Min.	Ringer	◁	Doubly diluted serum	▷	Ringer
March 10, 1937	0–30	−5		0		0
	30–60	0		10		0
	60–120	0		10		0

	* *	Ringer	◁	Doubly diluted serum	▷	Ringer
March 12, 1937	0–30	−5		45		0
	30–60	0		20		5
	60–90	0		10		15
	90–120	25		0		5

		Doubly diluted serum	▷	Ringer	◁	Doubly diluted serum
March 16, 1937	0–30	−10		−5		−10
	30–60	0		0		5
	60–90	−5		0		5
	90–120	5		0		10
	120–150	10		5		10

	* * *	Doubly diluted serum	▷	Ringer	◁	Doubly diluted serum
April 6, 1937	0–30	2		10		2
	30–60	0		5		0
	60–75	0		5		3

		Ringer	◁	Doubly diluted serum	▷	Ringer
April 8, 1937	0–30	15		5		20
	30–60	20		0		25
	60–95	25		5		30
	95–120	5		0		10

		Ringer	◁	Doubly diluted serum	▷	Ringer
April 8, 1937	0–15	0		0		0
	15–30	0		0		5
	30–45	0		10		2
	45–60	5		5		3
	60–95	0		15		40
	95–135	15		20		15

	* * * *	Ringer	‖	†Ringer	◁	Doubly diluted serum
April 12, 1937	0–30	−10		0		20
	30–45	−5		0		10

* The readings were begun 1½ hour late.
* * The readings were begun 1 hour late.
* * * The readings were begun ½ hour late.
* * * * The readings were begun 1 hour late.
‖ † Represents an impermeable wall.

As this usually happened at a later stage in the experiments we could not regard it as a sign of diffusion but as a result of the destruction of the cornea, possibly autolysis. This time, approximately the third hour of the experiments, *i.e.*, the fifth-sixth hour after excision is, according to other workers, about the limit of viability. We accordingly limited the time of our experiments. Our results show that under the influence of osmotic pressure the diffusion of liquid is not only possible in the epithelial-endothelial direction but also in the opposite manner.

We followed the same procedure in our experiments with chloride ions with the difference that we did not use a colloidal solution, but used variously diluted Ringer's solutions in contact with the two surfaces of the cornea and made our continuous chloride ion determinations with these solutions. For each determination we took 0·2c.cm. solution, added 4c.cm. distilled water and 2c.cm. of dilute nitric acid and by further adding 4c.cm. N/100 silver nitrate solution titrated the remaining Ag ions with N/100 NH_4SCN solution. The indicator ferri alum was added in larger quantities for with a greater indictator concentration the point of colour change is much more clearly defined.

In these experiments various concentrations of chloride ions were brought into contact with the epithelial and endothelial surfaces. The data show the appreciable chloride ion changes which resulted.

TABLE IV

Diffusion of NaCl through the cornea. The convex surface of the sign corresponds to the epithelial surface.

	Time H.	Time M.	per cent.		per cent.		per cent.
July 27, 1937	10	00	0·87	D	0·49	D	0·89
	11	30	0·89		0·50		0·88
	13	10	0·86		0·51		0·87
August 3, 1937	9	20	0·88	D	0·63	D	0·88
	11	00	0·87		0·65		0·88
	12	15	0·86		0·64		0·88
	12	50	0·88		0·64		0·88
August 3, 1937	9	20	0·88	D	0·70	D	0·89
	11	00	0·86		0·70		0·87
	12	15	0·88		0·68		0·89
August 5, 1937	10	00	0·92	D	0·33	D	0·93
	11	15	0·89		0·33		0·91
	12	20	0·90		0·36		0·87
	12	50	0·88		0·35		0·85

August 5, 1937	10	00	0·93	C	0·32	D	0·92
	11	15	0·92		0·34		0·92
	12	20	0·91		0·34		0·92
	12	50	0·89		0·34		0·90
August 9, 1937	9	00	0·89	C	0·66	D	0·88
	10	15	0·89		0·65		0·89
	13	15	0·88		0·68		0·86
August 9, 1937	9	00	0·89	C	0·70	D	0·89
	10	15	0·90		0·72		0·90
	13	15	0·89		0·72		0·88
August 13, 1937	10	10	0·89	C	0·88	D	0·89
	11	15	0·89		0·87		0·88
	12	20	0·88		0·87		0·88
August 13, 1937	10	10	0·87	C	0·87	D	0·88
	11	15	0·88		0·88		0·89
	12	20	0·89		0·88		0·88

That this is not a change as a result of evaporation (the chambers were not covered) is apparent from the fact that there were also decreases in concentration. Our results were undoubtedly influenced by the water diffusion due to the osmotic pressure difference, but we are unable to believe that along with this chloride ion diffusion did not also occur; for the reason that to induce the concentration changes that we noticed, 150-300c.mm. of water must have diffused, but this figure is far larger than the results we obtained for liquid diffusion in the above mentioned experiments. It is reasonable therefore to attribute the chloride ion concentration changes to an actual chloride ion diffusion.

We used in our experiments solutions in which the ion proportion corresponded to that of the Ringer's solutions. The chloride ion concentration in contact with the epithelial surface expressed in NaCl was 0·88-0·90 per cent. Variously diluted Ringer's solutions were brought into contact with the endothelial surface.

When the NaCl concentration of the solution in contact with the endothelium is between 0·88 to 0·60 (the epithelial concentration being 0·88-0·9 per cent.) diffusion can hardly be shown; but if the concentration is decreased below this figure, the changes become gradually much more noticeable, and the dilute solution becomes concentrated, the concentrated solution becomes dilute. This result expresses a great deal. It shows for instance that increase in the concentration of the endothelial solution runs parallel to the chloride ion decrease in the epithelial solution.

This fact would not have occurred had this diffusion taken place between the solution and the corneal tissue, for in that case the 0·88 per cent. solution in contact with the epithelium would behave in the same manner independent of whether a 0·90 per cent. or a 0·30 per cent. solution was in contact with the endothelium. The fact, therefore, that the concentration of the solution in contact with the epithelium can be influenced by the concentration of the solution in contact with the endothelium, of itself is proof that the forces responsible for the diffusion are not located in the cornea, in other words the living cornea behaves as an inactive permeable membrane. These results agree completely with the results obtained in our experiments with the diffusion of water.

These results are not only applicable to the " outside-in " direction, but also for the endothelial-epithelial direction. This was shown by our experiments in which the solution in contact with the endothelium was the 0·88 per cent. and various concentrations were used in contact with the epithelium.

There is evidence that a much smaller concentration difference will induce a measureable diffusion in this direction.

TABLE V

Diffusion of NaCl through the cornea. The convex surface of the sign corresponds to the epithelial surface.

	Time H.	Time M.					
July 26, 1937	10	15	0·57	◁	0·88	▷	0·54
	11	15	0·55		0·84		0·53
	17	10	0·55		0·84		0·55
July 26, 1937	10	15	0·53	◁	0·88	▷	0·52
	11	15	0·53		0·86		0·52
	14	10	0·54		0·82		0·53
July 30, 1937	9	45	0·88	▷	0·34	◁	0·90
			0·89		0·34		0·88
	11	45	0·86		0·35		0·86
			0·85		0·36		0·88
	12	45	0·85		0·36		0·85
			0·85		0·37		0·86
August 11, 1937	10	15	0·87	▷	0·63	◁	0·86
	12	10	0·87		0·65		0·87
	13	00	0·85		0·66		0·86
August 11, 1937	10	15	0·87	▷	0·63	◁	0·86
	12	10	0·86		0·66		0·86
	13	00	0·85		0·66		0·86

Summary

Our experiments dealing with diffusion through the excised cornea gave the following results:

1. Under the influence of colloidal osmotic forces a flow of water through the cornea is possible.

2. This permeability exists not only in the epithelial-endothelial direction, but also in the reverse direction.

3. Chloride ions under the influence of osmotic pressure can diffuse to the opposite surface in both directions.

NEW DEVELOPMENTS IN THE PHARMACOLOGICAL TREATMENT OF PRIMARY GLAUCOMA

BY

MARK J. SCHOENBERG, M.D.

NEW YORK

Introduction

IT is surprising that so little emphasis has been placed upon the significance of the fact that the pharmacological treatment of glaucoma consists, almost exclusively, of local applications of drugs which act principally upon the two subdivisions of the autonomic nervous system *in the eye*, the parasympathetic and sympathetic. This is probably one of the two reasons why the understanding of the mode of action of sympathomimetics (Sy-Mi) and parasympathomimetics (Pa-Sy-Mi)* upon the eyes has made little progress during past decades.

The other reason for this stagnation is that certain physiological data were missing. Without the knowledge of these data, no advance could be made. Recently, thanks to the theory of the chemical mediation of nerve impulses, the much-needed information has finally begun to become available. It has taken thirty

* Local applications of pilocarpine, eserine and of the new synthetic products, such as acetylcholine, mecholyl, prostigmin and doryl, have a stimulating action upon the ocular parasympathetic system and miosis is only one of several effects produced by these drugs. Atropine has an inhibitory or paralyzing effect upon the ocular parasympathetic system and mydriasis is only one of several reactions of the eye to this drug. It is, therefore, preferable to use the term, *para-sympatho-mimetics* (abbreviated: Pa-Sy-Mi), instead of miotics or mydriatics, when speaking of these substances.

The same reasoning is applicable to drugs which stimulate, inhibit or paralyze the ocular sympathetic. They should be called *sympatho-mimetics* (abbreviated: Sy-Mi).

years to lay the foundation of this new chapter of physiology and, although it is far from complete, it has already had a profound influence on our understanding of the physiology of the autonomic nervous system.

The object of this paper is twofold: (1) to submit some ideas concerning the mode of action of Pa-Sy-Mi and Sy-Mi in normal and glaucomatous eyes; these ideas are based upon data discovered with the aid of the theory of chemical mediation of nerve impulses; (2) to suggest a programme of work still needed to fill in some of the gaps in our knowledge of the pharmacological treatment of glaucoma.

Before taking up this subject, it is necessary to make a short review of:

The Essentials of the Theory of Chemical Mediation of Nerve Impulses*

For a long time physiologists have been wondering how impulses travelling along nerves are being transmitted from the nerve terminals to secretory or muscle cells and by virtue of what device does the nerve impulse bring about the activity of a secretory or muscle cell. A beginning of the answer was given by Elliot in 1905.[1] He observed that a smooth muscle which contracts when the corresponding sympathetic nerve is stimulated, will also act after the complete degeneration of the nerve (artificially induced by extirpation or section), if epinephrin is applied to that muscle. Since the epinephrin could not act on the degenerated nerve terminals, it follows that its action was limited only to the muscle cells. Elliot concluded that impulses travelling along sympathetic nerves do not act directly upon the smooth muscle cells by " jumping " the neuromuscular partition. He

* The pharmacological studies which have uncovered data on chemical mediation of nerve impulses are numerous and it would be needless to review them in this paper. Those desiring to familiarize themselves with the subject will find an extensive bibliography in the papers of:

BUTT, H. R.—Chemical Mediation of Nerve Impulses. *Arch. Neurol. and Psychiat.*, Vol. XXXVII, p. 142 (January), 1937.

MYERSON, A., and THAU, W.—Human Autonomic Physiology. IX. Effect of Cholinergic and Adrenergic Drugs on the Eye. *Arch. of Ophthal.*, Vol. LXXV, p. 78 (July), 1937.

DALE, SIR HENRY.—Transmission of Nervous Effects by Acetylcholine (Harvey Lectures, July, 1937). *Bull. N. Y. Acad. of Med.*, Vol. XIII, p. 380 (July), 1937.

LOEWI, OTTO.—The Humoral Transmission of Nervous Impulse. (Harvey Lectures, 1932-1933; pp. 218-233.)

NEWTON, W. H.—Chemical Transmission at Nerve Endings. Recent Advances in Physiology, pp. 284-328, 1936.

CANNON, W. B.—Autonomous Neuro-effector Systems. New York, 1933.

1. ELLIOT, T. R.—The Action of Adrenalin. *Jl. of Physiol.*, Vol. XXXII, pp. 401-467, 1905.

assumed that on the arrival of the impulse at the nerve terminals an epinephrin-like substance (Sy) is " secreted " and that this chemical substance acts as the stimulus, bringing about the function of the muscle cells. *This chemical substance is the chemical mediator of the sympathetic nerve impulses.* Since 1905 a large number and a great variety of experiments were performed by physiologists and Elliot's experiments and conclusions were confirmed. The study of the transmission of nerve impulses to motile and secretory cells has been extended to the two components of the autonomic nervous system (the sympathetic and parasympathetic) and the fundamental discovery has been made—namely, that nerve impulses are transmitted to the secretory or contractile cells by the aid of a chemical mediator. The most important findings can be summarised as follows:

1. Impulses reaching the parasympathetic nerve terminals stimulate the formation of an acetylcholine-like substance (A-C), which acts upon the corresponding motile or secretory cells (effector cells).

2. Nerve impulses reaching the sympathetic nerve terminals release an epinephrin-like substance, called sympathin (Sy) which mediates the action of the impulse upon the corresponding effector cells.

3. The source of the chemical substance present at the neuro-effector junction from which the nerve impulse creates A-C is dependent for its maintenance on the integrity of the nerve endings. This source disappears or becomes depleted when the nerve fibres degenerate.

4. The A-C is destroyed by an esterase very soon after it has been produced. It is only during a short time, before it is neutralised, that A-C acts upon the effector cells. The process is repeated rhythmically as long as the nerve impulses arrive.

5. Eserine has the property of inhibiting the neutralising effect of esterase upon A-C.

6. Atropine prevents the effect of A-C.

7. Ergotoxin and ergotamin (gynergen) inhibit or paralyse the stimulating action of Sy on the effector cells.*

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* Velhagen has demonstrated in the aqueous and tissue of the iris and retina a substance possessing all the properties of acetylcholine. He found that this substance acts directly on the sphincter muscle of the iris.

VELHAGEN, K., Jr.—Cholin-Acetycholine as Physiologic Components of Retinal and Uveal Tissue. *Arch. f. Augenheilk.*, Vol. CV, p. 573, 1932.

The Action of the Cholines on the Muscles of the Iris with Special Reference to their Double Innervation. *Ibid.*, Vol. CVIII, p. 126, 1933.

Further Experiments on Active Principles in the Ocular Tissues. *Ibid.*, Vol. CIX, p. 195, 1935.

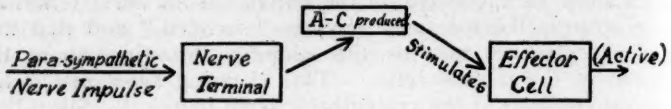


Fig. 1.a. Para-Sy neuro-effector unit in action.

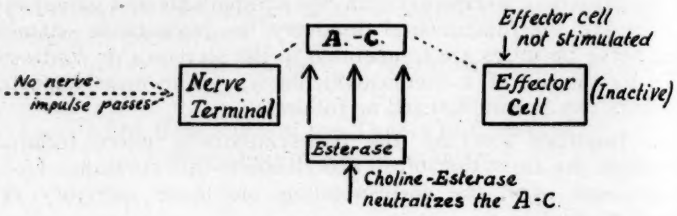


Fig. 1.b. Para-Sy neuro-effector unit inactive

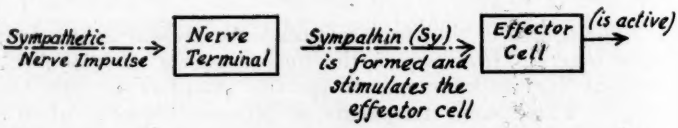


Fig. 2. Sympathetic neuro-effector unit in action.

We must keep in mind constantly that *we are dealing with a unit, consisting of nerve terminals, effector cells, and the neuro-effector junction.*

The implications of this theory are that the normal performances (release of A-C, esterase, contraction, relaxation, secretion) of the neuro-effector unit (setting the effector cells into action) depend on the following factors:

(*a*) the normal condition of the nerve fibres and nerve terminals;
(*b*) the " secretion " or release of normal amounts of A-C, or sympathin, respectively;
(*c*) the state of receptivity of the effector cells;
(*d*) the release at the parasympathetic nerve terminals of a normal amount of esteraze which neutralises the surplus of A-C. (No similar substance has been found for sympathin.)

One may readily understand that in the eye, in which the two branches of the autonomic system are richly represented, a pathological process of the nerve fibres or their terminals, an increase or diminution of secretion of the chemical mediator (A-C or Sy), a reduction or an increase of sensitivity of the effector cells; or an increase or decrease in the quantity of esterase (at the nerve terminals) will bring about a disturbance of the function of the tissues innervated by one or of both branches of the autonomic nervous system.

The Mode of Action of Pa-Sy-Mi in the Eye

Having in mind the essential principles of the theory of chemical mediation of nerve impulses and the implications derived from them, we can better understand the mode of action of " mimetics " in the eye.

The Pa-Sy-Mi

The instillation in the eye of any one of the drugs, like —pilocarpine, eserine, prostigmin, acetylcholine and its derivatives (mecholyl and doryl)—has in many respects a similar effect: the pupillary and ciliary muscles contract, there is a relaxation of the zonula Zinii and, under favourable circumstances in glaucoma, the ocular tension is reduced. But, each one of these substances acts on a definite part of the neuro-effector unit.

Acetylcholine and its more stable derivates (mecholyl and doryl) are chemically quite similar to the A-C-like substance, which mediates the transmission of the parasympathetic nerve impulse. They act directly upon the effector cells just as if a nerve impulse, travelling along the nerve has released the chemical mediator (A-C). *They act upon the effector cells even in the absence of a nerve impulse or when the intensity of the impulse is diminished.*

The action of *eserine* and *prostigmin* is localised at the neuro-effector junction. It prevents the esterase from neutralising the A-C and, thereby, gives it a chance to act upon the effector cells (motile and secretory). Eserine cannot act directly upon the effector cells if no A-C is released.

Pilocarpine stimulates the effector cells, if they are chemically receptive.

In other words, the secretion of the ciliary processes and the contraction of the pupil and of the ciliary muscle, the change in the patency of blood vessels may be produced artificially by chemical substances which increase the amount of A-C, inhibit the action of esterase or stimulate directly the effector cells. These substances also increase the accommodation and tend to reduce the ocular tension.

' *Scopolamine, atropine,* and *homatropine* have an inhibitory paralysing effect, especially by *rendering the corresponding effector cells insensitive to A-C.* Practically, no matter how much eserine or A-C may be used in a thoroughly atropinised eye, the pupil will not contract nor will the ciliary body perform its function.

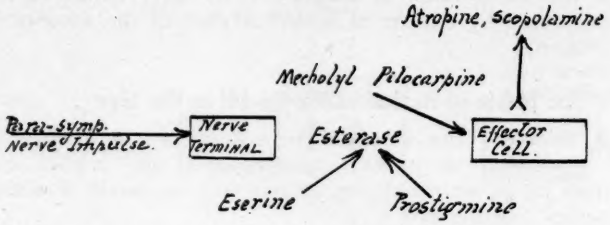


Fig 3 Seat of action of mecholyl, pilo., eserine and prostigmine, atropine, scopolamine.

The Si-Mi

Epinephrin, ephedrin, benzedrin, ergotoxin or ergotamin tartrate act upon the sympathetic neuro-effector unit.

Epinephrin acts directly upon the effector cells. Its effect is similar to sympathin. It increases the size of the pupil, decreases slightly the accommodation, tends to raise the ocular tension and contracts blood vessels.

Cocaine is a vasoconstrictor and a dilator pupillae. It increases considerably the effect of epinephrin. Its action is on the sympathetic nerve terminals (?).

Ephedrine and *benzedrin* act somewhat similarly to epinephrin.

Ergotoxin and ergotamin tartrate (gynergen) inhibit the specific affinity of the effector cells for Sy. Their action on these cells is similar to that of atropine on the para-sympathetic effector cells.

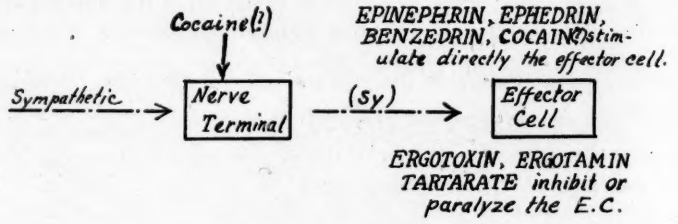


Fig 4 - Seat of action of epinephrin, ephedrin, benzedrin, cocain, and ergotoxin

The Rationale of the Pharmacological Local Treatment of Primary Glaucoma

The rationale of the pharmacological treatment of glaucoma is based upon the assumption that the appearance of glaucoma signs and symptoms is in a *certain measure* related to a disturbance of the function of the autonomic nervous system *in the eye*. Some of the circumstantial evidence which speaks in favour of this assumption, one can enumerate:

(*a*) the effective action of some Pa-Sy-Mi and Sy-Mi (with miotic action) in reducing the ocular hypertension and minimising other manifestations of glaucoma;

(*b*) the disastrous effect of some Pa-Sy-Mi and Sy-Mi (with mydriatic and cycloplegic action) in glaucoma, both groups of drugs being essentially Pa-Sy-Mi, respectively Sy-Mi;*

(*c*) the specific response of the pupils to the instillation of epinephrin in eyes with prodromal glaucoma (the Knapp reaction);

(*d*) the fact that Pa-Sy-Mi do not reduce the ocular hypertension when administered by mouth or parenterally (in non-toxic doses), but diminish it when applied directly to the eye.

These evidences and the facts established with the aid of the theory of chemical mediation of nerve impulses, sketched in the first part of this paper, justify the conclusion that *in glaucoma, one seat of disturbance is located in the neuro-effector unit of the autonomic nervous system within the eye.*

Thus, we are led to think that at least for the present, in treating a case of primary glaucoma by pharmacological means, the first thing to do is to attempt to re-establish the normal function of the autonomic neuro-effector units within the eye.† Though groping in the dark, empiricism has done this very thing. It is significant that two drugs—eserine and pilocarpine—used against glaucoma during the past six decades, have a selective action upon the neuro-effector unit of the ocular autonomic nervous system. The recent additions to the pharmacological armamentarium against glaucoma (acetylcholine group, prostigmin and ergotoxin group), also have a specific affinity for and action upon the neuro-effector units.

Is our pharmacological armamentarium against primary glaucoma complete?

---

* One must admit that other changes besides blocking of the drainage system take place in the eye after using mydriatics and cycloplegics.

† This should not be interpreted to mean that these units are the only factor or the most important one involved in the causation of glaucoma. One can only say that besides surgery, the vegetative neuro-effector unit is, at the present time, the most accessible and responsive factor to our therapeutic efforts.

Looking over the lists of drugs we realise its deficiencies and we are enabled—perhaps for the first time—to formulate the specific need for other drugs which:

(1) *Stimulate or recondition the nerve endings of the parasympathetic fibres;*

(2) *Re-establish normal chemical processes that supply A-C;*

(3) *Reduce the over-production of esterase;*

(4) *Enhance the receptivity of the effector cells;*

(5) *Reduce the production of Sy, and*

(6) *Diminish the over-sensitivity of the sympathetic effector cells.**

The problems are both clinical and pharmacological. It is probable that the diversity of responses of the glaucomatous eye to certain drugs depends in no small measure upon the location of the disturbance (whether predominantly in the parasympathetic or in the sympathetic; whether in the nerve terminals in the acetylcholine or sympathin formation area, or in the effector link of the neuro-effector unit. In the future, it will be the clinician's task to indicate whether the types are predominantly sympathetic, parasympathetic or mixed, and to point out which link or links of the neuro-effector units are involved in the various types or stages of the disease.

Indications

Pilocarpine and eserine, the drugs commonly employed in glaucoma, are indiscriminately used at present, either alone or in combination, without a definite indication. Since eserine prevents the neutralising effect of esterase on A-C and since it has no direct action upon the effector cells, it should be used in cases in which the amount of A-C is released in too small quantities or is being reduced by the over-production of esterase. Pilocarpine, on the other hand, acting directly upon the effector cells, is indicated when there is a deficiency in the function of these cells. Although we are as yet unable to distinguish between these two theoretical groups, the attempt has been made to separate them. (Case reports will be subject of a later communication.)

In cases in which there is a functional preponderance of the sympathetic component of the vegetative nervous system (with over-production of sympathin) or an enhanced sensitivity of the effector cells to Sy, drugs like ergotoxin or ergotamin tartrate,

* Walter B. Cannon (*Proceedings of the Staff Meetings of the Mayo Clinic*, Vol. XIII, January 19, 1938) has recently made the following statement: "It would appear that there may be a general law that denervation *increases* the sensitiveness of structures to stimulation." This statement is based upon a number of reliable experimental data.

which have a depressing or paralysing effect upon the sympathetic effector cells, are indicated. In such cases, A-C, pilocarpine and eserine may act as an adjuvant, but cannot succeed alone in correcting the condition.

In mixed forms, such as a failing parasympathetic and an over-acting sympathetic, drugs that enhance the function of the first and depress that of the second have a better chance of success.

Summary

1. Nerve impulses travelling along the two branches of the vegetative nervous system are transmitted to the secretory or contractile cells by the aid of a chemical mediator (acetylcholine or sympathin, respectively).

2. There are evidences which support the assumption that in primary glaucoma, there is a disturbance (functional or organic) of one or both branches of the vegetative nervous system, within the eye.

3. The present pharmacological treatment of primary glaucoma endeavours to correct such a disturbance by dealing with each or with all three of the components of the neuro-effector unit.

4. As far as we know, drugs that have a beneficial effect upon eyes with primary glaucoma, possess the property of acting either on the esterase (which neutralises acetylcholine) or on the effector cells.

5. There is need of more knowledge on the subject of dysfunction or degeneration of the parasympathetic and sympathetic nerve terminals, more information on details regarding the process of release of acetylcholine and esterase, also on affections of effector cells. There is also need of drugs or other means which can re-establish the normal function of any one of the three components of the neuro-effector unit, whenever impaired.

6. Clinical experience leads us to believe that in some cases of primary glaucoma, we are dealing with a failing function of the ocular parasympathetic, in others with a preponderance of the ocular sympathetic, and in still other cases with a combination of both conditions. Thus, we may classify—for the present—the various types of primary glaucoma in three groups: sympathetic, parasympathetic and mixed.

7. The adoption of this classification may prove to be helpful to the progress of our knowledge of the pharmacological treatment of primary glaucoma.

Thanks are due to the firms of Hoffman La Roche, Sandoz and Merck for furnishing prostigmin, gynergen and mecholyl for trial and study.

BINOCULAR FIXATION FOR SCOTOMETRY AT ONE METRE

BY

J. FOSTER

LEEDS

SCOTOMETRY at 33 cm. while recognised as less accurate, and as being a less delicate test than at one or two metres, possessed for a long time the compensating advantages of binocular fixation and rapid recording. In the last few years the speed of recording at greater distances than this has been increased by various devices. Amongst the latter one may mention Tudor Thomas' projection device, Affleck Greeves' stitched screen, and the Foster protractor.

Binocular fixation at longer distances is still rather a problem, however, and existing apparatus is either apt to interfere with the field of the examined eye, to be somewhat inaccurate, or complicated and expensive.

Cowan and Markov (*Amer. Jl. Ophthal.*, 1929, p. 656) have produced a simple method which overcomes these difficulties, but their device is still rather unwieldy.

The device described below incorporates portions of the design of several existing methods of binocular fixation. It allows for a long working distance, non-interference with the field of the tested eye, a simple check on the accuracy of fixation, and is relatively simple in practice and construction.

Two models are described; the first being more suitable where illumination on the scotoma screen is variable, and where a chin rest (to which the apparatus can be fastened) is employed.

The second model (which is the one I employ personally) is more convenient where a chin rest is not used, and the illumination of the Bjerrum screen a constant quantity. The instrument consists of two parts:

1. *Circular Target.*—This is a grey circle of 5in. external diameter, painted on a 7in. square of black paper, which is pinned to the Bjerrum screen so that the centre of the circle overlies and conceals the fixation point on the screen, in the same way as its precursor the Fincham target.

The circle has four inward projections like the stubs of the spokes of a wheel. The screen should have an even surface illumination of at least 7ft. candles—as poor illumination of the target renders it invisible through the second part of the instrument.

2. *The Cross Projector.*—This resembles an ordinary ophthalmoscope, with a battery handle containing a resistance.

The patient, seated at one metre, holds the instrument in one hand, and looks through the sight hole through a half-silvered mirror, which is sloping at an angle of 45° towards, instead of away from, the user, as in an ordinary ophthalmoscope.

A movement of the switch projects the illuminated image of a cross on to this mirror—which appears as if it were situated on the Bjerrum screen.

The resemblance to an ophthalmoscope is very strong, and the head of the device can be adapted to any self-illuminated ophthalmoscope.

When it is desired to make a scotometric examination of a patient (at one metre) with a central scotoma in his left eye, it may prove impossible to obtain central fixation for the eye, and the target may be entirely or partially invisible. If he looks through the unilluminated fixation device with the other eye, the whole of the target will be visible. (Fig. 1.)

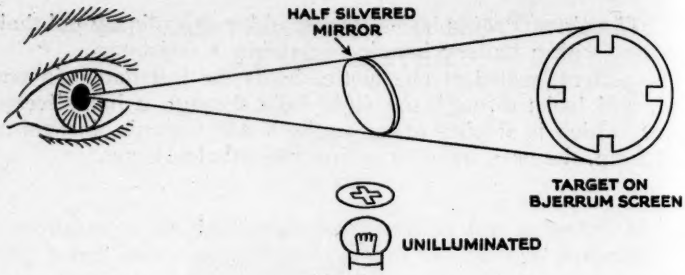


FIG. 1.

The device is now switched on at a low voltage. The target and an illuminated cross are simultaneously visible, and he can then centre the latter to fuse with the inward projections of the former, by moving the battery handle which he holds (Fig. 2) in front of the right eye.

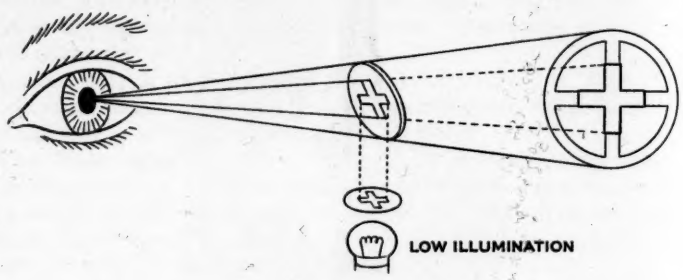


FIG. 2.

A. The illumination is increased by turning the switch, and the target gradually disappears from view of the good eye, leaving only the cross, which the patient fixes. (Fig 3.)

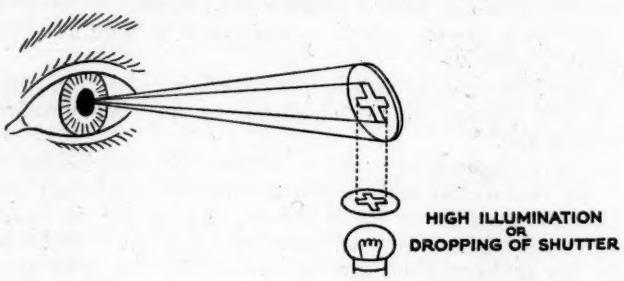


FIG. 3.

Scotometric examination of the left eye is now carried out, and the fixation checked occasionally by reducing the illumination in the fixation device to make sure that the cross is still centred in the target.

B. If enough of the target is still visible to the bad eye to allow of fusion symmetrically with the cross, stages one and two may be omitted.

If a normal person centres the cross by method " A " with his right eye, and keeps the other closed, the cross will appear to shift a little horizontally from the centre of the circle when the left eye is re-opened. Similarly, if the fully illuminated cross is fused with the target, and illumination then reduced, it will be found to be displaced in the opposite direction. This phenomenon is due to heterophoria, and when it can be estimated, as in a normal person, a compensating trial case prism can be placed in the groove behind the sight hole of the instrument.

As, however, in actual practice, central fixation is completely lost in most of the cases one wishes to test, and the heterophoria probably does not vary throughout the examination, this slight error can be neglected.

Model II

I, personally, do not employ a chin rest for scotometry, and it was found in practice that certain patients held the instrument rather unsteadily, so that moving the resistance moved the instrument a little. If, therefore, one has a constant illumination on a scotoma screen, and the device is not fixed to a chin rest or made firm in any other way, another modification of it may prove more convenient in use, and it is in this form that the device was finally made up.

The resistance is replaced by a switch, and the silvering on the mirror is removed so that at maximum illumination it is still possible to see the circle and the cross simultaneously with the good eye. A small shutter is dropped over the sighting hole between the screen and the silvered glass, producing the same effect as increasing the illumination. The shutter can be raised and lowered very easily from either side of the patient to check whether the cross is still in the circle or not.

The diagnostic devices described in this article are all produced by Messrs. Rayner and Co., 100, New Bond Street, London, W.1, for whose assistance in their development I am greatly obliged.

NOTES ON OPTICAL INSTRUMENTS AND DEVICES

BY

J. FOSTER

LEEDS

OPHTHALMIC NEEDLE HOLDER

THE NEEDLE HOLDER illustrated is a combination of the handle and scissors shank of Gillies' needle holder, and of the jaws of Quarry Silcock's ophthalmic needle holder. It is extremely useful,

and adds to the speed of ophthalmic suturing in all situations, if the " forceps' tie " method is employed : this is specially marked in lid operations. The scissors portion is most useful except for dividing sutures near the inner canthus where there is not enough room to employ it. The instrument is made by Down Bros.

THE ARTIFICIAL GLAUCOMA HALO

OCCASIONALLY one suspects a patient of having had an attack of sub-acute glaucoma. It is, however, difficult to explain to some of them exactly what a glaucoma halo looks like. A great deal of prolonged and futile description is saved if the disc (which consists of a layer of lycopodium powder between two thin sheets of glass affixed in a trial lens rim) is placed in a trial frame. Attention is directed to a Maddox spot light, and an extremely good reproduction of a glaucoma halo thus produced. A definite " yes or no " can be elicited at once.

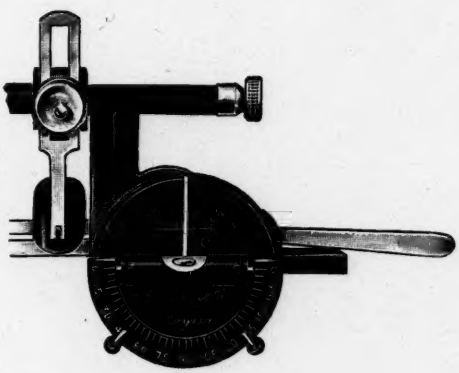
Occasionally one encounters patients who claim that the reproduction is more, and others who claim that it is less vivid than their own halo. Very occasionally patients can see no colour in the artificial halo at all. In practice, however, I find the agreement is very close. This phenomenon was first discovered by

NOTE.—These have been shown at R.S.M. meetings and some have been published in opticians' advertisements.

Fraunhofer, and its clinical application suggested by Elliot. This device reduces the test to a convenient form for clinical use. No illustration is appended as the device is indistinguishable from an ordinary trial-case lens.

TRIAL FRAME LEVEL

CERTAIN patients are unusually sensitive to small changes of cylinder axis position. This may be due either to the strength of the cylinder required or extremely good perceptive judgment. One also encounters patients in whom there is a considerable amount of vertical asymmetry of the face and eyes, with resultant tilting of the trial frame. A small curved bubble level filled with



glycerine to reduce the rapidity of the bubble excursion is attached to a metal disc of the same size as an astigmatic trial lens. The horizontal line on the disc is arranged to coincide with the horizontal marks of the frame, and a constant or momentary deviation of the bubble from the centre of the level will indicate an error which must be corrected either by tilting the head or adjusting the cells or sides of the frame.

THE THREE-LENS TEST

As the effective diameter of the modern trial lens is 20 mms., it is unnecessary to employ lenses of a larger size in an end-point test; it is possible, therefore, to combine a concave sphere, a

similar convex sphere, and a cross cylinder in an extremely small space and thereby to accelerate ease and speed of handling.

RETINOSCOPY RACK

IN principle the retinoscopy rack is as well known as the device described above as the three-lens test. Racks are available from 15in.—4ft. in length, and from 1in.—4in. in breadth. Since the pupil seldom exceeds 6 mms. in diameter, and as all text-books are careful to point out, only the central portion of this is considered in retinoscopy, it is unnecessary to employ lenses of a greater diameter than this figure.

Adhering to this principle, it is possible to incorporate in a retinoscopy rack which will fit in the waistcoat pocket the lenses required for the great majority of refractions. Twenty-four lenses ranging from ± 0·5 to ± 6·0 in 0·5D intervals are fitted into an aluminium strip, 16 cm. long, 2 cm. wide, and 0·25 cm. thick. For rapid reading by the light reflected by the retinoscopy mirror, the strength of the lenses is marked in the interspaces, the concave side of the rack coloured red, and the convex black.

In use the rack is extremely rapid, and is so light and small that it causes no unsteadiness when held at arm's length. For cases requiring lenses of a greater strength than 6D. I prefer to use separate lenses, but at the suggestion of J. Benson, of Ediburgh, a cursor can be fitted to extend the range to ± 12·0. The rack has

the failing of all racks, namely that retinoscopy is performed at the distance of half a metre, and that one must be careful to have no other source of illumination behind the user, or the small lenses will be obscured by the reflex. Other users have complained that

there is too much reflection from the lenses not in use. This difficulty is easily overcome by a slight movement of the hand holding the rack. The rack is so arranged in the latest models that the hand holding it can be rested against the forehead; the weaker lenses are, in consequence, at the end of the rack nearer the hand.

ANNOTATION

Surgical Narcosis

Recently there has been some discussion on the production of what has been termed surgical narcosis for major ophthalmic operations by the administration of drugs which will lessen or remove the distressing psychological effects associated with the operation. It is claimed by some that the mental shock of an ophthalmic operation to elderly persons is the cause of post-operative delirium, flatulence, abdominal pain and absence of peristalsis.

It is obviously desirable that the drugs used for the purpose of inducing narcosis should not cause restlessness, sneezing, jactitation and other disagreeable features which might jeopardise the satisfactory conduct of the operation or spoil the eye in the period immediately following it. Of the drugs used some produce a tranquil, acquiescent state with retention of consciousness and others render the patient unconscious. Some require the assistance of inhalation anaesthesia. Sedative drugs such as morphia, omnopon, scopolamine, paraldehyde and the barbiturate preparations such as sodium evipan, sodium pentothal, nembutal and avertin have been tried.

Morphia, omnopon and scopolamine given hypodermically are satisfactory in some cases in which a tranquil and passive state is induced without loss of co-operation. However, some persons have an idiosyncrasy to opium derivatives and post-operative vomiting may be a serious source of danger. Flatulence, abdominal discomfort and restlessness are other features which make these drugs undesirable for use in intra-ocular surgery. Of the drugs which produce unconsciousness paraldehyde administered by the rectum has attracted recent attention. In some cases it has caused post-operative muzziness which lasts for two days, restlessness, vomiting and delirium, complications which it is unjustifiable to risk in ophthalmic surgery.

Avertin per rectum dispenses with the patient's co-operation; the conjunctiva becomes pale, the pupil small, the eye fixed in the middle position and the conjunctival and corneal reflexes are absent. This drug also lowers the blood pressure and intra-ocular pressure and occasionally it causes post-operative vomiting and restlessness. Another disadvantage is that it has often to be used in combination with a general anaesthetic. Sodium evipan is administered intravenously and will render the patient anaesthetic for 20 minutes or so. It is necessary to use a local anaesthetic as well, and in some cases this drug causes violent sneezing, twitchings, raising of the head and jactitation, incidents that would frustrate an ophthalmic operation or lead to disaster.

Pentothal sodium, an intravenous anaesthetic, has a narrower margin of safety than sodium evipan but it does not induce such complications as twitching, sneezing and jactitation. The induction is quick, anaesthesia is quiet and unperturbed and the administration of this drug may be used up to one hour. After operation recovery of consciousness is uneventful, there is no restlessness or other undesirable consequence. The intra-ocular pressure falls and the eye is fixed in the central position. Pentothal sodium is, we think, the best of the intra-venous anaesthetics for ophthalmic use.

Nembutal in many cases is satisfactory for preparing a patient

for operation. If the dose is too heavy the patient becomes semi-conscious, restless and unable to co-operate. Its effect is variable but when it works well it is admirable.

The Ophthalmological Society of Australia

Early in the month of April of this year the newly founded Ophthalmological Society of Australia was formally decided on in Sydney. Until now, while there have been ophthalmic sections of the British Medical Association in Queensland and Victoria and an eye and ear section in South Australia, together with an Ophthalmological Society of New South Wales, there has been no federal body to embrace the whole Commonwealth, and Western Australia and Tasmania have had no ophthalmic provision at all.

The new Society starts with a membership of 110 and its first President is Sir James Barrett. We feel confident that Australia could not have chosen better for the first of what we hope will be a long line of distinguished Presidents. Sir James Barrett was a student of Lord Lister, a fellow research worker with William Lang and he has been a member of the Ophthalmological Society of the United Kingdom for close on half a century.

Committees on various aspects of ophthalmology such as (*a*) literature and publications, (*b*) research and scientific, (*c*) orthoptic training, (*d*) industrial injuries and compensation, have been set up. Among the conveners are Mr. Bruce Hamilton and Dr. Ringland Anderson.

It is hoped to have the first general meeting in Melbourne in March, 1939. Transactions will be published and the Society hopes later to be in a position to undertake the publication of an ophthalmic journal.

It is with great pleasure that we learn of this new undertaking; nothing is more likely to stimulate research work in Australia and we wish it many years of successful enterprise.

ABSTRACTS

I.—RETINA

(1) **Arruga, H. (Barcelona).—Treatment of detachment of the retina.** *Arch. of Ophthal.*, October, 1937.

(1) After paying a justifiable tribute to the pioneer work of Gonin, who was "learned, sincere and good," **Arruga** deplores the fact that the treatment of retinal detachment is not as good as

it should be. The principal reason is that owing to the diversity of clinical types, the operator must adapt his measures according to the conditions in each case. Accurate ophthalmoscopic examination and localization of the hole are the keynote of success, and although a large and extensive operation is frequently indispensable, the desideratum is to sacrifice the smallest possible area of retina. The examination should be carried out under full mydriasis (aided by the subconjunctival injection of adrenalin) by both the direct and indirect method and the results entered on one of the recognised forms of chart. Ophthalmoscopic examination during the operation is almost indispensable, and can be used to assist localization in several ways:—

(1) Diathermic puncture is performed with a needle through the suspected area and the position of the resulting white mark with reference to the retinal hole is noted with the ophthalmoscope. If the retina is too bulging to have been touched by the needle, some of the sub-retinal fluid is allowed to escape with the aid of pressure on the eyeball and the procedure repeated, when the white area will usually be visible. If this is unsuccessful a small needle or knife may be introduced, which will then be visible.

(2) Transillumination, the operator views the tear with his ophthalmoscope, and the assistant marks on the sclera the portion which is illuminated from within the eye. Alternatively the light is applied to the sclera and is moved about until the operator sees it illuminating the tear. In applying surface diathermy a current of 30-60 milliampères is recommended; for perforation, the strength should be 80-150. In the latter case, the perforations should be separated by at least 0·75 mm. or inconvenient scarring will result. In surface coagulation, the applications may be made closer together. Vortex veins should of course be avoided. It is important in perforating diathermy that the punctures should remain open for some time. When there is much fluid the needles should be 0·5 mm. in diameter or failing this, the perforating action should be prolonged and the borders of the hole enlarged, galvano-cautery may also be used.

One of the secrets of success is that the retina should lie in contact with the choroid at the conclusion of the operation. With regard to post-operative treatment, the author considers that in general, 6-8 days in bed are sufficient, the dressing being changed every three days and the binocular bandage kept on for 12 to 20 days, after which stenopaeic glasses are worn. So far as pathogenesis is concerned Arruga considers that adhesions between the framework of the vitreous and the retina are the principal cause of the tears.

F. A. W-N.

(2) **Fuchs, A. (Vienna).—On the importance and aetiology of holes in detachment of the retina. (Sur l'importance et l'étiologie des trous dans le decollement de la retine).** *Arch. d'Ophtal.*, Tome 2, No. 1, p. 18, 1938.

(2) **Fuchs** points out that the condition to which he refers is one of interest not merely to ophthalmologists but to all medical men in the widest sense, and adds that whereas formerly, the majority of those suffering from detachment of the retina became permanently blind, now, largely due to the genius of Gonin, the greater number can be cured.

Separation of the retina is not in itself a disease, but arises from various causes, either traumatic or idiopathic, the latter term being principally associated with myopia and senility. The trauma may be of a relatively trifling nature and a long time may elapse between the date of the causative injury and the recognition of the actual detachment.

Combinations of various factors may make it very difficult to determine exactly the group into which certain cases fall.

As regards anatomical changes two come into particular prominence, (1) the hole in the retina, and (2) retraction of the vitreous body. The former is much the more important from both the clinical and therapeutic point of view. It was Gonin who emphasized the importance of exact localization of the tear or hole in the retina, and obtained such wonderful results by closing it by means of the thermo-cautery. The satisfactory closing of the holes enables re-attachment of the retina to be permanent. The second condition preliminary to retinal detachment is the presumed presence of a liquid in the vitreous body.

The vitreous is a colloid which can vary its composition, and is capable of producing a large amount of fluid.

The vitreous body being itself detached may pull the retina away from its bed, and by the production of excessive fluid, which may escape through the hole in the retina, may cause its further upheaval.

The vitreous body is only fixed, normally, near the ora serrata and its detachment usually commences in the upper part, with the result that the very weight of the body augments the detachment. Such detachments of the vitreous are frequent in myopes, particularly senile myopes.

It is only in a small proportion of cases of retinal detachment that no holes are to be found and the author emphasizes the need for distinguishing between holes, and tears.

The former are round, oval, or polygonal, and may be present in numbers, or perhaps only one may be observed. Sometimes the "lid" of a hole may be seen floating in the vitreous in front of it.

Tears on the other hand, may appear as slits, or in horse-shoe shape, and many show ragged edges, which are usually rather

unduly pale. The long narrow tears may escape detection through being concealed by the folds of the detached retina.

Another group of tears is to be found at the furthest forward point of observation: these latter give a good prognosis when dealt with by means of diathermy or katholysis.

These tears too, are of interest from a pathogenetic point of view as they are frequently the result of trifling injuries, and yet they may be of unlimited extent—sometimes, as in the case of injuries at birth, being so extensive as to lead to an erroneous diagnosis of glioma. The situation of the tears is of importance and figures show that after a contusion the detachments are usually in the upper and outer part. When the retinal detachment commences at the ora serrata they are usually in the lower temporal quadrant.

The round holes have no particular predilection as regards situation and may be found anywhere, perhaps most frequently in the upper and outer quadrant.

As to the causes of retinal tears and ruptures, they are many but two theories predominate, (1) Traction caused by the vitreous body, and (2) Primary cystoid degeneration of the retina. Fuchs supports the former and does not think that the advocates of the second have made out their case except in regard to a very limited number of a special class.

Congenital fragility of the retina may also play a part, but the author thinks this constitutes no more than merely a weakness which tends towards the formation of a detachment if other aetiological causes present themselves. He considers that weakness of the retina should cause no more surprise than should the cases of undue susceptibility of the cornea in certain subjects, to take on disproportionately serious features, a state of affairs probably due to some fragility of the membrana limitans of Bowman.

Tension of the retina should not be under-estimated as an aetiological factor in cases of extreme myopia as, though perhaps in itself insufficient to cause a hole, it may in combination with other conditions play an important rôle in their origin through traction on the vitreous, malnutrition, etc.

Cysts of the retina too, may cause tears—frequently bilateral and in the lower temporal sector. Fuchs recommends that operative measures in these cases should not be too hastily undertaken, as they tend to recover spontaneously.

Holes near the macula are frequently unobserved until the macular region is itself invaded by the detachment, and if the holes in the periphery are satisfactorily closed the retina is able to re-attach itself completely.

Besides the causes he discusses, Fuchs emphasizes that there are many others of great diversity in both their origin and in their

effects. He maintains that there can be no hard and fast laws laid down in this aspect and guidance is only to be obtained by means of careful observation combined with unbiassed judgment.

F. R. HILL.

(3) **Venco (Pavia).—Holes at the macula. (Perforazione traumatica della macula lutea).** *Rass. Ital. d'Ottal.*, May-June, 1937.

(3) After a short dissertation on the various forms that injury to the retina may take, **Venco** discusses the special lesion of macular holes. He recounts a case of a motorist who was struck violently while driving by a stone thrown up by a wheel. The direction of the blow seemed to have been directly from before backward. There was immediate loss of vision. When the hyphaema which followed allowed examination of the fundus, a typical hole was seen. Round the hole the retina was slightly raised and oedematous.

The author draws attention to the fact that holes of the retina at the macula may be caused by agencies other than injury; Ogilvie, in his paper read to the Ophthalmological Society in July, 1900, was inclined to lay too much stress on injury as the cause; perhaps the present reviewer was in part responsible for this, as the case which he quoted to Ogilvie (no. x in the paper) was more probably an instance of atrophy of the macula, but was included as traumatic at that time.

HAROLD GRIMSDALE.

(4) **Nastri (Rome).—The causation of idiopathic detachment of the retina. (Contributo allo studio della genesi di distacco idiopatico della retina).** *Rass. Ital. d'Ottal.*, July-August, 1937.

(4) There is at present no agreement as to the cause of rupture of the retina, though all are agreed upon the necessity of closing tears if cure of detachment is to be obtained. **Nastri** has had the opportunity of following the course of the onset of detachment in three patients, and the interesting observations then made are here recorded.

In two cases, the first things noted were slight oedema of a region of the retina with small haemorrhages; in this region a tear was seen later and a detachment. In the third case, there was already a tear but the surrounding retina was oedematous, and there had been muscae, which perhaps suggest previous haemorrhages.

In the author's opinion, the course of things is this: by slow changes, the retina has acquired adhesion to the vitreous. The pull on these adhesions caused by rotatory movements of the eye, acting on some part of the retina already degenerated, causes rupture of small vessels with consequent haemorrhages, and the retina tears in the affected region; the degeneration of the retina must precede any other change.

HAROLD GRIMSDALE.

BOOK NOTICES

Text-book of Ophthalmology. By SIR W. STEWART DUKE-ELDER. Vol. I. Second impression, with corrections and additions. London: Henry Kimpton. 1938. Price, 63s.

It is a matter for congratulation that so advanced and highly scientific a treatise on the development, form and function of the visual apparatus as is contained in the first volume of Sir Stewart Duke-Elder's Text-book should so have appealed to ophthalmologists that a second impression has been demanded in a space of six years. The author has seized the opportunity "to bring up to date certain sections wherein fundamental advances in knowledge have rendered the original text misleading, such as, for example, in the sections dealing with ocular pigment and the neurogenic origin of pigmented tumours, the biochemistry of the eye, the nature of the intra-ocular fluids, the physico-chemistry of the vitreous body, the chemical and electrical reactions in the retina, and so on."

Perhaps the most important change is a modification of the author's views on the formation of the intra-ocular fluid. Many readers of the first edition must have felt that there were well authenticated arguments against so dogmatic a view that the intra-ocular fluid was a simple dialysate. It is to be noted that this theory is now modified "by the interposition of a membrane with distinct physiological properties lining the chamber of the eye." The author is to be commended for his courage in definitely stating his present opinion that "as in most questions in physiology, an end has by no means been reached in the questions discussed here: in fact, subsequent work has shown that in the first edition of this text-book an impression of greater finality than was justified was conveyed to the reader. The estimation of minute quantities of organic materials is always a difficult matter, and when these are bound up in the complexity of the body-fluids the difficulties are multiplied exceedingly. In many cases, indeed, chemistry is by no means sufficiently far advanced to provide the means to make dependable analyses and pragmatic conclusions possible, and as time goes on more elaborate methods of technique will produce more accurate results necessitating a further revision of ideas, and advances in knowledge will indicate pitfalls as yet unsuspected. This evolution, of course, is the greatest fascination of the biological sciences, and those who are not prepared to modify a hypothesis to suit new facts (which will certainly appear) cannot hope to enter into the spirit of physiology. Actually recent advances in biochemistry have shown that many of the figures quoted in this section will require in future to be modified."

The Practice of Refraction. By SIR W. STEWART DUKE-ELDER. Third Edition. Pp. 340, Figs. 183. Published by J. and A. Churchill, Ltd. 1938.

The third edition of this useful book is on the same lines as its predecessors, but contains additional matter dealing with new work. A short description of aneisokonia and its diagnosis is given but as the author wisely says, " it is yet too early to give a dogmatic estimate of its clinical importance." Contact glasses receive notice too, and a concise account is given of the methods evolved by Dallos for fitting and making them. The author has been misinformed, however, when he states that " the necessity of any fluid between the cornea and the glass is abolished," because one of the features of the Dallos glass is that the inner corneal surface is ground to a spherical shape. In all but exceptional cases therefore, fluid is required though not usually in such quantity as in the older types of glass. It is a pity also that in describing the disadvantages of contact glasses, he should say that there are many people who seem unable to wear them, because in actual practice it is remarkable how few these are when adequate care has been taken to secure a good fit and to train the patient in the technique of inserting and wearing the glasses.

Dynamic retinoscopy and orthoptic training are also described in sufficient detail for the refractionist to assess their value.

Those who are familiar with the earlier editions of this volume will realise that the value of a useful book has been further enhanced by the production of this, its third edition.

Introduction to Ophthalmology. By PETER C. KRONFELD, Professor of Ophthalmology, The Peiping Union Medical College. Pp. 299, Illustrated. Baltimore: Charles Thomas. Price, 3.50 dollars.

The author has set out to present the principles of ophthalmology which form a necessary part of medical education and it has been his purpose that this book should form a supplement to the short and essential practical course to which the teaching of ophthalmology has been reduced in many medical schools.

The text is concerned mainly with pathological principles and details of diagnosis, methods of examination and treatment have been omitted as the author thinks that these are better imparted by oral instruction and demonstration.

The chapters discussing the pathology of uveitis, the physiology of the retinal circulation and the intra-ocular pressure and its pathological variations will be particularly interesting to the post-graduate but are probably too advanced for the undergraduate.

There are five plates reproduced in monochrome illustrating fundus disorders. Plate I, picture 1, labelled the normal fundus

of the right eye, illustrates the left fundus and shows a temporal crescent and the perimacular region is suspiciously pathological. Some of the other drawings are poor.

There is an index and ophthalmological dictionary. Certain common ophthalmic terms which have not been mentioned in the text are placed in appropriate alphabetical order in the index and receive a few lines of explanation and description.

Transactions of the American Ophthalmological Society. Vol. XXXV. 1937.

The 73rd Annual Meeting of the American Ophthalmological Society was held at Hot Springs, Virginia, on June 3, 4 and 5, 1937, under the Presidency of Dr. Harry Friedenwald.

The volume contains many papers of scientific and clinical interest, some of which we hope to publish later in abstract form in the *British Journal of Ophthalmology*. The quality of these original papers, the production of the illustrations and the printing is up to the usual high standard of this work.

The volume also contains a list of the officers and council, past Presidents and members of the Society.

CORRESPONDENCE

SMOOTH MUSCLE OF THE PERIORBITA AND THE MECHANISM OF EXOPHTHALMOS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—I have read with interest Dr. Brunton's account of the periorbital unstriped muscle and his remarks concerning the possible part played by its contraction in the mechanism of proptosis as found in thyrotoxicosis. It may be that this muscle has some slight influence in the direction stated, but I think it would be unwise to attribute to it anything more in that respect than just that of a coincidental nature, for the following reasons:—

1. It is stated that contraction of the unstriped periorbital muscle produces exophthalmos by causing an increase of retro-bulbar pressure. This is not likely to be the case in Graves' disease because the periorbital fascia is fairly closely attached to the walls of the orbit and any contraction would not produce more than a slight rise in the retro-bulbar pressure. If the periorbital muscular layer were able to free itself from this attachment as a result of its

continued spasm, thereby achieving an efficient increase in the retro-bulbar pressure, the space so developed would surely form a constant feature at autopsy.

2. In the typical case of thyrotoxicosis with exophthalmos there is not the slightest sign of increased retro-bulbar pressure.

3. Contraction of the periorbital membrane in dogs, cats, sheep, etc., produces marked exophthalmos because, (*a*) it is a well developed muscular membrane, (*b*) it is in greater part a free and mobile structure. Waves of peristalsis may be seen to pass freely along it following a stimulation of the cervical sympathetic. In the ape, however, in which the orbit takes the form of a bony socket and the periorbital membrane is attached all round, thus resembling the human type, stimulation of the cervical sympathetic does not produce a protrusion of the eye-ball. Hence it may be pointed out that it is hardly logical to expect protrusion of the eye-ball in man in these circumstances.

Finally it may be added that the type of exophthalmos seen in Graves' disease is typically associated with lid retraction, a factor which undoubtedly depends upon a conjoint mechanism.

I am, etc.,

J. H. MULVANY.
(*Radium Registrar*)

KING'S COLLEGE HOSPITAL,
S.E.5, *21st May, 1938.*

CORNEO-SCLERAL SUTURE IN CATARACT EXTRACTION

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—I have read with interest Mr. H. B. Stallard's article on "A Corneo-scleral Suture in Cataract Extraction, etc." in the May number of the Journal.

I have used a suture consistently for some time in all my cases of cataract extraction and agree with him as to the value of a suture of some kind. The suture which he describes is corneo-scleral—I very much prefer a purely conjunctival suture, a full description of which will be found in the *Trans. Ophthal. Soc. U.K.*, Vol. LIII, p. 86, 1933.

This suture, like the corneo-scleral, is inserted before the section is made, and the technique of its insertion presents no difficulties. Its chief advantages, in my opinion, over the corneo-scleral suture are that a generous conjunctival flap can be obtained and that, owing to the very considerable tension exerted by the tightened conjunctiva, the lips of the wound are held in such close apposition that the danger of prolapse, short of direct violence to the eye, is

practically abolished. In all uncomplicated cases I perform a simple extraction, and have never seen a prolapse in a case of extra-capsular extraction when this suture has been inserted.

I cannot agree with Mr. Stallard's statement that "stitches in the conjunctival flap are useless" in checking loss of vitreous. On the contrary, the tension exerted by this conjunctival suture is so effective that in case of vitreous loss the iris repositor can if necessary be inserted between the lips of the wound without further flow of vitreous after the suture has been tied. With regard to its effectiveness in preventing post-operative hyphaema, I am not qualified to speak, as this is a complication with which I have fortunately not been troubled.

I am, Yours truly,

R. AFFLECK GREEVES.

23, WIMPOLE STREET, W.1
May 27, 1938.

NOTES

Honour

IN the Birthday Honours List we are pleased to see the name of Wing-Commander P. C. Livingston, who receives the O.B.E.

* * * *

British Medical Association, Section of Ophthalmology

AT the annual meeting of the British Medical Association in Plymouth this month the following are the officers of the section of ophthalmology. President: Sir Stewart Duke-Elder; Vice-Presidents: R. E. Bickerton, E. H. Cameron, C. B. F. Tivy; Hon. Secretaries: R. W. Paine (Plymouth), Wing-Commander P. C. Livingston.

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Ophthalmological Society of Egypt

THE new Council of the Ophthalmological Society of Egypt has been formed for the year 1938 as follows:—President: Dr. Mohammed Mahfouz Bey, Principal Medical Officer, Ophthalmic Section, Government Hospital, Alexandria; Vice-President: Dr. Hassan Barrada Bey, Ophthalmic Inspector, Ministry of Public Health, Cairo; Hon. Treasurer and Archivist: Dr. Eisa Hamdi El Maziny Bey, Director, Ophthalmic Hospitals, Ministry of Public Health, Cairo; Hon. Secretary: Dr. Mohammed Khalil, Ophthalmic Surgeon, Qalawoon Hospital, Cairo; Hon. Assistant Secretary: Dr. Ahmed Abdel Rehim Fahmy, Tutor, Ophthalmic Hospital, Giza.

All communications are to be addressed to the Hon. Secretary, Ophthalmological Society of Egypt, 4, Baehler Street, Cairo.

Chengtu Eye, Ear, Nose and Throat Society

THE Chengtu Eye, Ear, Nose and Throat Society, the first society in China representing jointly those practising in ophthalmology and oto-laryngology, was organized at the Eye, Ear, Nose and Throat Hospital at Chengtu, West China, on December 14, 1937. The following officers were elected:—President: Eugene Chan, M.D.; Vice-President: Robert A. Peterson, M.D.; Secretary: C. C. Teng, M.D.

The Society will meet at the Chengtu Eye, Ear, Nose and Throat Hospital.

The members of this Society are derived from the Departments of Ophthalmology and Oto-laryngology of the West China Union University of Chengtu, West China, of the National Medical College of Nanking and of Cheeloo University of Tsinan, Shantung, and from practitioners specializing in these fields in Chengtu and other centres in West China. The transfer of medical schools with their faculties and students from the war-zone in East China to Chengtu in West China has created here a unique centre of medical teaching and clinical activity. The personnel from the Departments of Ophthalmology and Oto-laryngology so transferred are co-operating in the undergraduate and graduate teaching and clinical activities of the Departments of Ophthalmology and Oto-laryngology of the West China Union University.

* * * *

Glass Eyes in 1681

THE following advertisement appeared in Merlin's *Ephemeris* of 1681:—

William Boyse, or Boys. At the sign of the Golden Griffin, in Belle Savage Yard on Ludgate-Hill, the only English operator in glass, and the most expert in making artificial eyes, so exact as not to be distinguished from Natural, they are of enamel, with colour mixt of the same, without either paint or lead, and worn with much ease, and so curious that they have the motion of the natural eye, being exactly made to the colour or bigness of the same which renders them very ornamental and commodious, the like was never made in England, the truth whereof is well known to many Persons both in our own and some other nations.

Wm. Boys died before the year 1710 and was succeeded by his son-in-law, James Smith.

* * * *

Société française d'ophtalmologie

LA Société Française d'Ophtalmologie held its annual congress in Paris from May 16 to May 19 at the Centre Marcelin-Berthelot. The British members received a warm welcome from the Secretary-

General, Dr. Onfray, and from the Treasurers, Dr. Bruneau and Dr. Charpentier.

The most important paper was that of Dr. Hambresin of Brussels on shock-treatment in ophthalmology; this had been printed and circulated beforehand. A cinematographic demonstration of an operation for cerebral tumour by the neurological surgeon, Dr. de Martel, exhibited surgical dexterity and photographic ingenuity at their highest pitch; it was worth a visit to Paris for this alone.

The traditional excursion by motor-coach enabled congressists to see over several chateaux of historic interest, as well as the moated grange of Villiers which has been inhabited ever since the thirteenth century; this latter is the centre of an agricultural estate and both are in the possession of our colleague, Dr. Velter. The annual dinner, which completed the tour, was held at the Trianon Hotel, Versailles.

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Oxford Ophthalmological Congress

WE gave a preliminary note of the proposed activities of the Oxford Congress on page 317. We would remind readers that the dates are July 7-9. Dr. Traquair will open a discussion on "Anaesthesia in ophthalmic surgery," and Professor Cairns will open the discussion on ocular palsies from the neurological standpoint.

Mr. Williamson-Noble will read a paper on "Contact glasses considered from the clinical standpoint, with a survey of results obtained." Dr. van Heuven, of Utrecht, will speak on "Papill-oedema," and Dr. Czukrász Ida, of Debrecen, will speak on "Hungarian plastic methods." Papers are announced by Mr. Harrison Butler, on "Capsular glaucoma," and by Mr. Frederick Ridley, on "An 'active' substance in the tears." Professor Lauber, of Warsaw, will speak on "The relationship between intracranial and retinal blood pressure and intra-ocular tension; the treatment of tabetic optic atrophy." Mr. Cole Marshall will demonstrate a film to illustrate operation for retinal detachment by diathermy and electrolysis.

FUTURE ARRANGEMENTS

1938

July 7-9.—Oxford Ophthalmological Congress, at Oxford.
October 7—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *May, 1938.*

HAGEDOORN. Cysts of the margin of the eyelid.
FREY. The rôle of transfusion in ophthalmology.
LAMB. The genesis of the cyclitic membrane.
PRANGEN and KOCH. Divergence insufficiency: a clinical study.
SPACKMAN. X-ray studies of the nasolacrymal duct.
LANGDON. Treatment of detachment of the retina by use of the thermophore.
JULIANELLE and HARRISON. Studies on the infectivity of trachoma.
BERENS. Tenon's capsule transplants in surgery of the ocular muscles, with especial reference to post-operative deviations with adhesions between the muscles and the eyeball.

Annales d'Oculistique. *May, 1938.*

MAGITOT. Glaucoma without hypertension.
WINCKLER. New researches on the morphology and the topography of certain elements in the orbital region.
VIALLEFONT and FUENTES. Leprosy in ophthalmology.
CERKEZ. Sclerotic fibroma.
KALASCHNIKOV. Eserine. Its use in the treatment of keratitis.

Archives d'Ophtalmologie. *May, 1938.*

JAYLE. An attempt at introduction to the study of the paths and the centres of the conjugate ocular movements.
DESVIGNES. A case of compression of the intra-cranial optic nerve.
FRANÇOIS. Familial haemorrhagic angiomatosis and its ocular complications.

Archiv für Ophthalmologie. *May, 1938.*

WIEDERSCHEIM. Miners' nystagmus in the Saar coal district.
KYRIELEIS. Investigations on dark adaptation with a new method of recording automatically threshold stimuli.
GILLESSEN. Experimental investigations on the question of the contractility of a lens fibre.
SEEFRIED. About vitamin C in senile cataract.
ZUBLER. The entoptic indication of the yellow colour of the macula centralis retinae.
GESERICK. Knowledge of endangiitis obliterans.
LOBECK. A new kind of use of the achromatic double lens in slit-lamp examinations.
SCHMIDT. Tuberculosis of the bulbar conjunctiva.
KAPUSCINSKI. The effect of external administration of vitamin A to rats' eyes and its influence through vitamin D.
KAPUSCINSKI. The influence of the intra-ocular tension in youthful forms of glaucoma.
THIES. Why operate early for severe ocular burns?
OHM. Nystagmus.
GOLDMANN and LIECHTI. Experimental research on the genesis of irradiation cataract.

Zeitschrift für Augenheilkunde. *April, 1938.*

DAVIDS. The question of the treatment of ocular tuberculosis.
SCHEYHING. Isolated paralysis of accommodation, after fracture of the base of the skull.
RINTELEN. Differential diagnosis of choroidal sarcoma.
JAKOBOVITS. Cord-formation in the anterior chamber after parenchymatous keratitis.

Klinische Monatsblätter für Augenheilkunde. *May, 1938.*

WERNCKE. Tay Sachs' familial amaurotic idiocy.
SCHMIERER. A case of ectogenous conjunctival tuberculosis.
STOCKMAN. The colour of the eye.
OLÁH. New observations on rosacea iritis.
CZUKRÁSZ. The operation of iridodialysis.
CZUKRÁSZ. Sarcoma of the lacrymal fossa, simulating a lacrymal sac tumour.
BRUNNER. X-ray representation of the lacrymal sac region and the technique of the endonasal lacrymal sac operation.
FAVALORO. Contino's anterior glaucoma.

Acta Ophthalmologica.

ROSENGREN. The treatment of retinal detachment by means of diathermy and injection of air into the vitreous.
MALLING. An investigation of glaucoma capsulare.
SJÖGREN. The knowledge of kerato-conjunctivitis sicca.
LUNDSGAARD. A Danish genealogical tree with hereditary transmission of Leber's optic atrophy.
VON BAHR. A case of uveo-parotitis with perivasculitis and other rare symptoms.
VON BAHR. A case of myoblastic myoma of the lacrymal sac.
LUNDBERG. New method of fixation to determine the visual field in a case of unilateral central scotoma.
ANDERSSON. Is a perforation of the globe necessary for the production of sympathetic ophthalmitis?
KARPE. An examination of the deep displacement of the second lens reflexes in accommodation.
UVNÄS and WOLFF. On the occurrence of acetyl-choline esterase in the aqueous humour and vitreous body.

Bollettino d'Oculistica. *March, 1938.*

CARAMAZZA. Experimental tuberculosis of the ocular globe from inoculation of human tubercle bacilli.
DE'CORI. Oscillations of the corneal curvature (astigmatismo pulsante).
BIETTI. Choroidal metastases from latent primary carcinoma and diagnostic problems that derive from it.
ALIQUO-MAZZEI. Ocular manifestations with tumours of the base of the skull of pharyngeal origin.
MUSCATELLO. On one singular histological aspect (metamorphosis?) of bilateral conjunctival hyperplasia in trachoma.

Rassegna Italiana d'Ottalmologia. *January-February, 1938.*

SGROSSO. Glioma of the optic nerve, clinical, anatomical, and pathological contribution.
GALLENGA. The ocular components of optic neuromyelitis (Devic's disease).
SIOTTO. Suppurative echinococcal cysts of the orbit.
BERTOLDI. The therapeutic efficacy of ether benzil-cinnamon in trachoma.
MATHIS. Research on the sensibility of the cornea to the virus of poliomyelitis.
DUC. Experimental research on the adhesive treatment of retinal detachment with fulguration.

Revista Oto-Neuro-Oftalmologica. *January-February, 1938.*

CARAMAZZA. Orbital phlegmon.
BIETTI. Considerations on the appearance of papilloedema in myopic eyes.
AGNELLO. Syndrome of nose and trigeminal neuralgia from traumatic corneal lesions.

Archivos de Oftalmologia de Buenos Aires. *February, 1938.*

FORTIN. The foveal mosaic.
DAMEL and VILLEGAS. Orbital cylindroma.
ORTIZ. The instruction and the professional orientation of amblyopes. Creation of special schools.
ALLENDE. Total extraction of the opaque crystalline lens by means of Snellen's method.